



May Allah enable me to get my  
ideas into practice

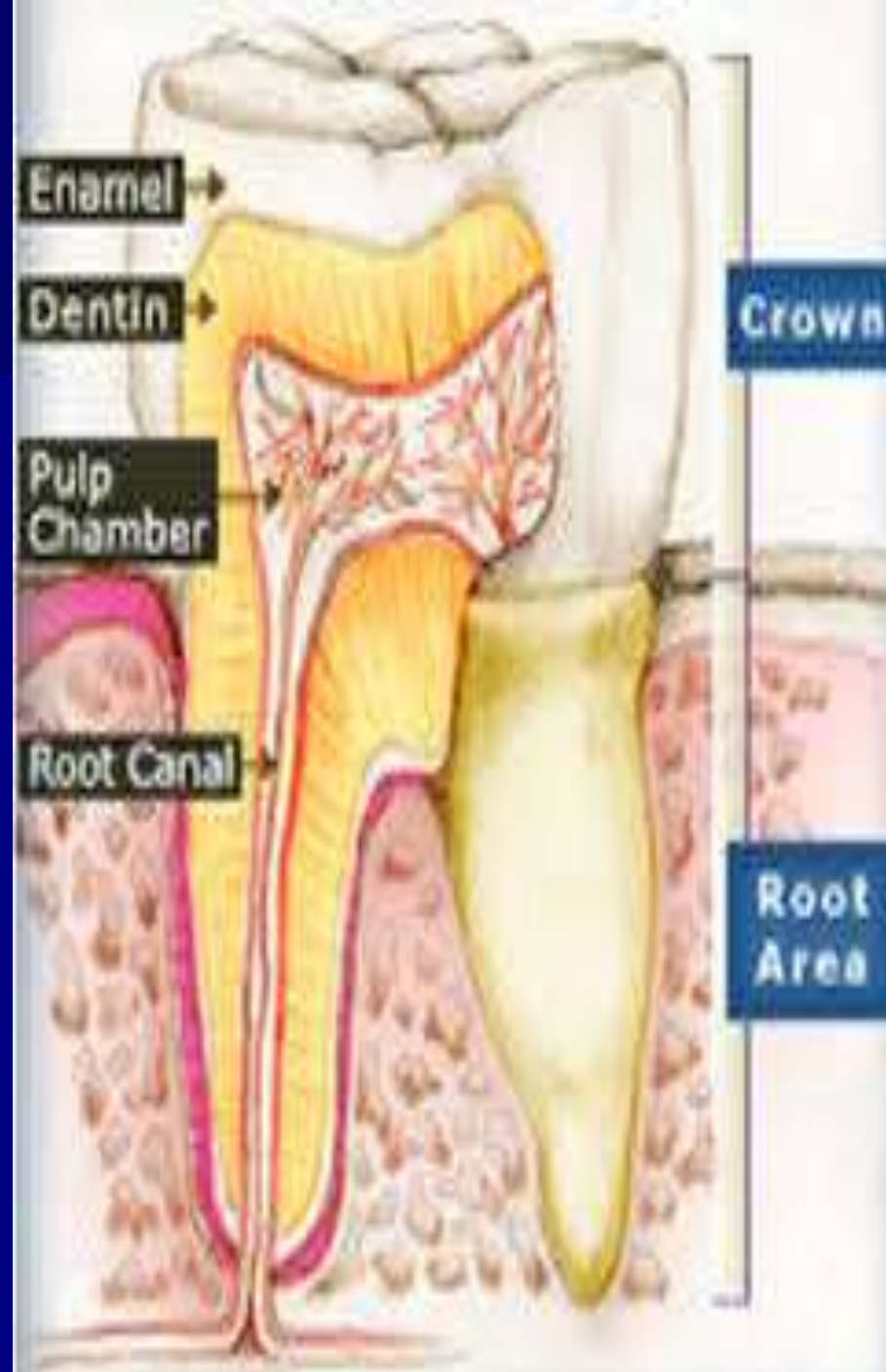
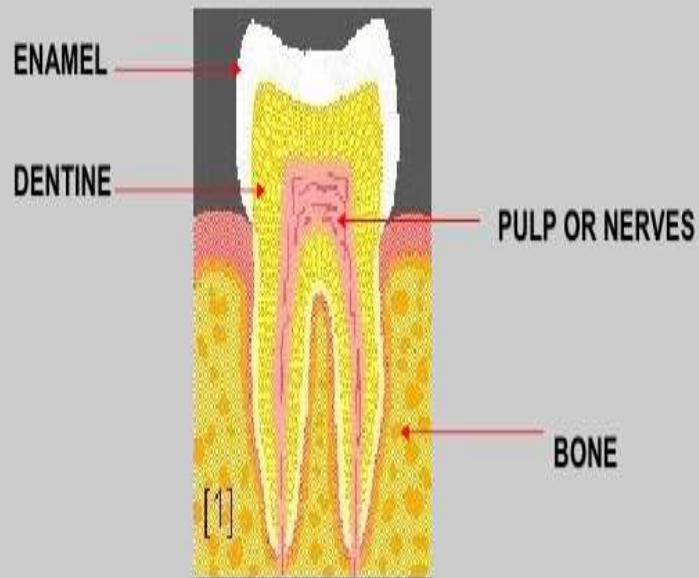


# Dental caries

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Faculty of Dentistry  
Mansoura University***



# What is tooth?



# Structure of your teeth



## **Enamel** (top)

The hard material on the outer surface of the tooth.

## **Dentin** (middle)

Below the enamel – the bonelike material that surrounds the sensitive inner parts of the tooth.

## **Pulp** (inside)

Tissue that contains nerve endings and blood vessels.

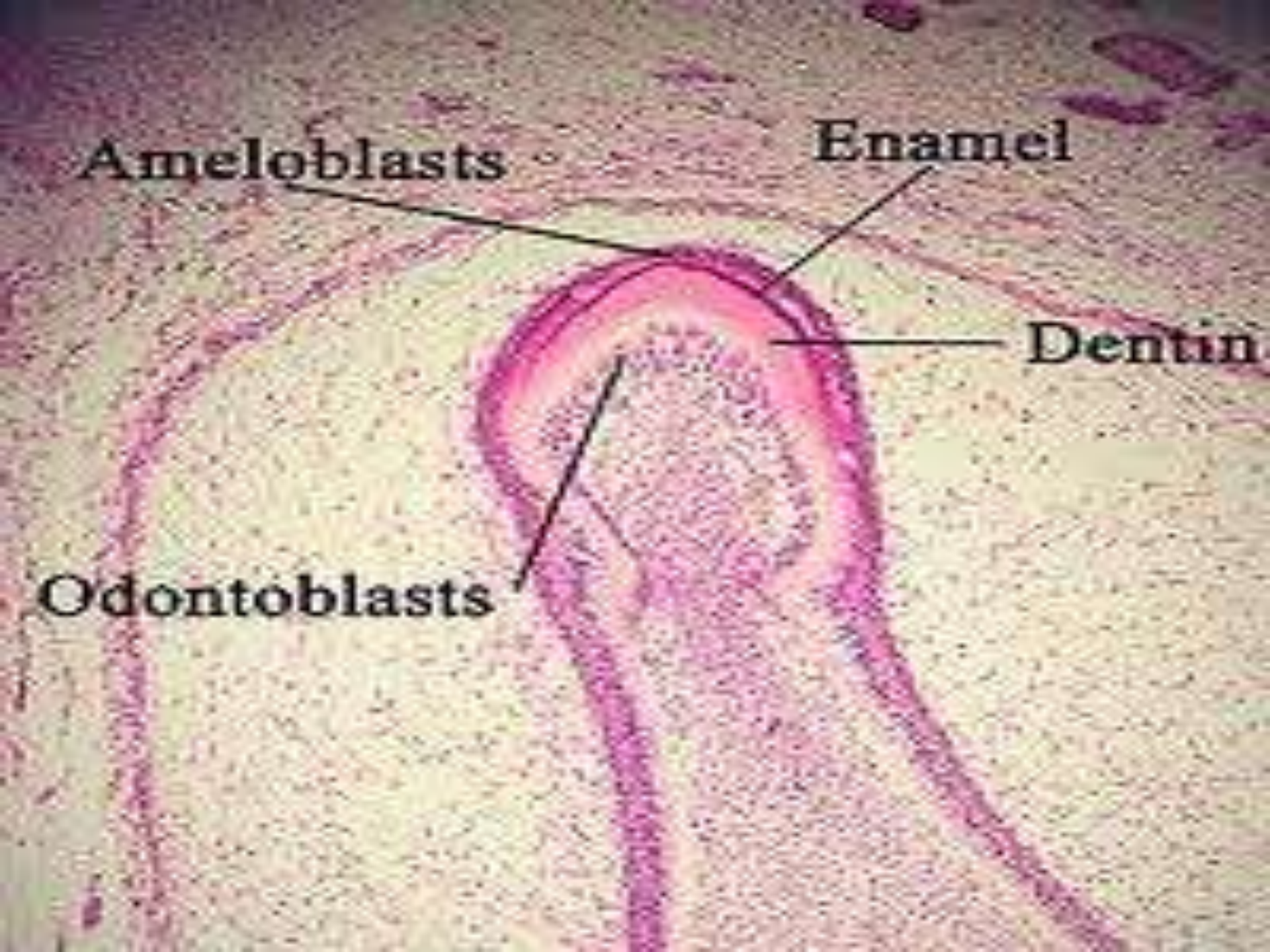


**Ameloblasts**

**Enamel**

**Dentin**

**Odontoblasts**



# What is Dental Caries?

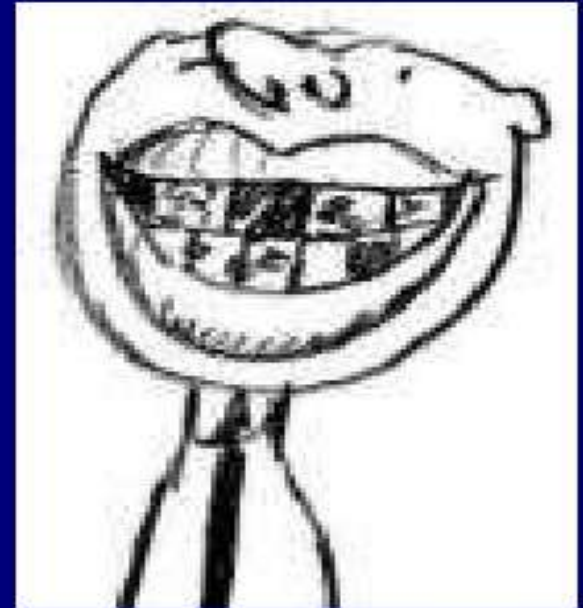


If we do not care properly for our teeth, we may get **Dental Caries**. Caries make holes in teeth caused by plaque. Often, caries is a very painful disease.



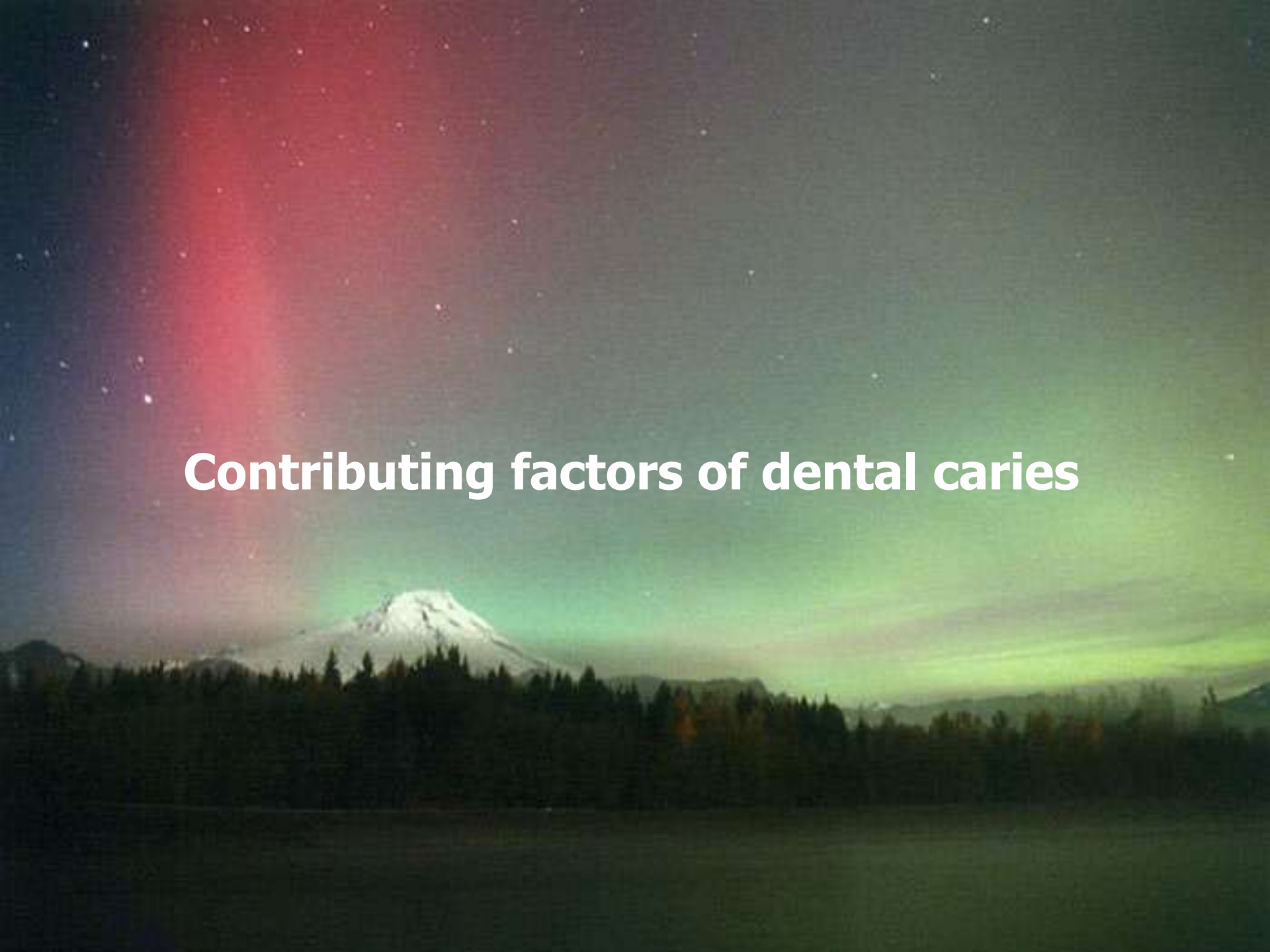
# DENTAL CARIES

- Progressive bacterial damage to teeth exposed to saliva.
  - one of the most major causes of all diseases and major cause of tooth loss.
  - ultimate effect-to breakdown enamel and dentin and open a path for bacteria to reach pulp.
- Consequences-inflammation of pulp and periapical tissues.



***Bacterial damage of the hard tooth structure  
(demineralization of inorganic and destruction of organic  
substance of the tooth)***

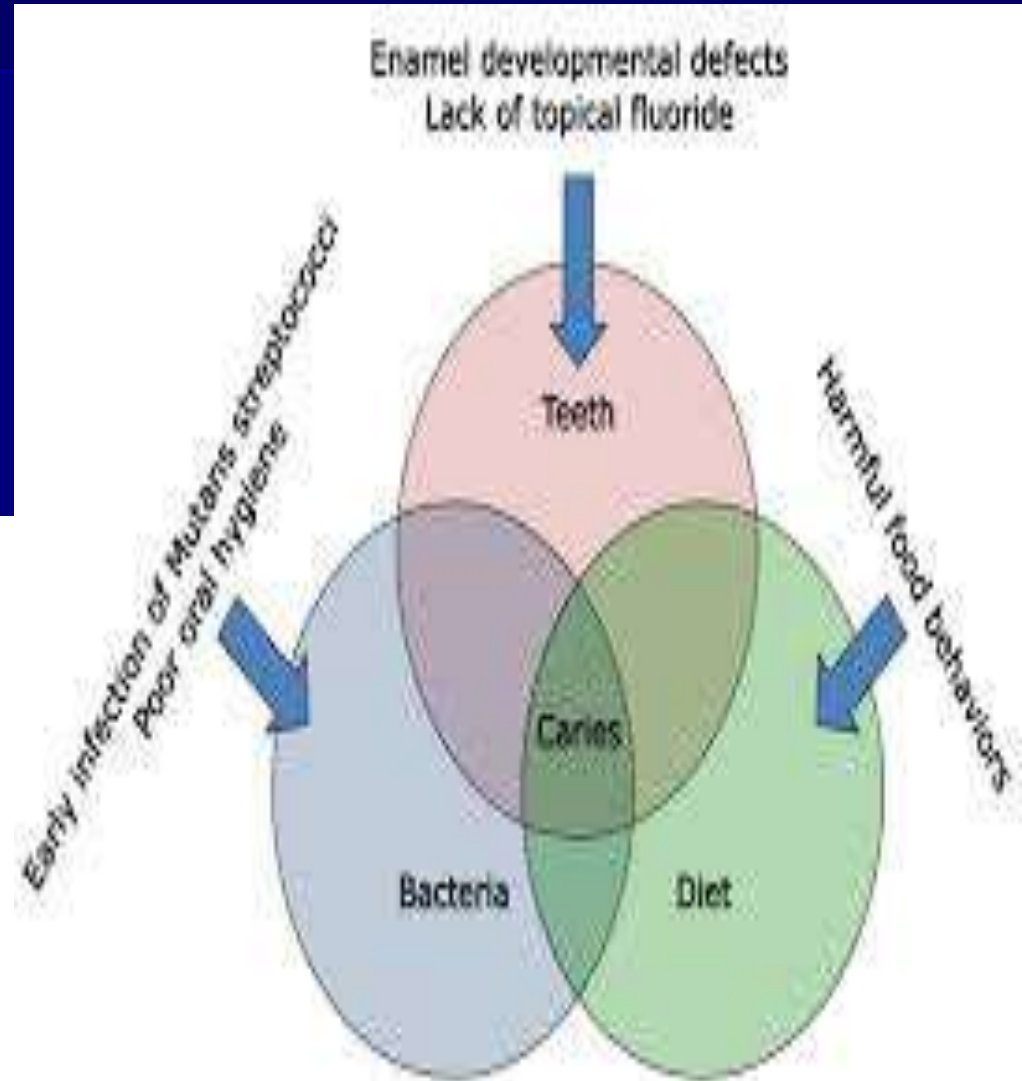
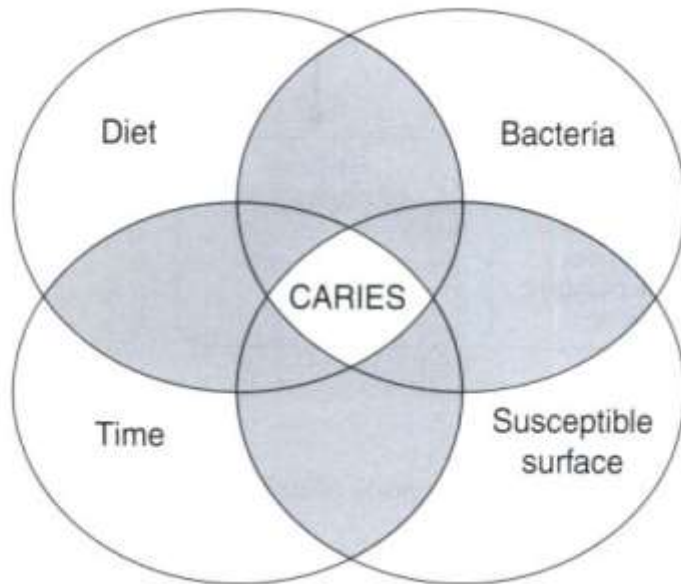
# Contributing factors of dental caries





# Contributing factors of dental caries

1. Susceptible tooth surface
2. Carbohydrate
3. Micro-organisms
4. Dental plaque



The background of the image is a solid black field. Overlaid on this is a complex, organic pattern of glowing particles. These particles are primarily orange and yellow, with some white highlights, and they form a dense, swirling, and somewhat chaotic shape that resembles a nebula or a microscopic view of a biological structure. The particles are concentrated in the center and spread out towards the edges, creating a sense of depth and movement.

**Susceptible tooth surface**



# Susceptible tooth surface

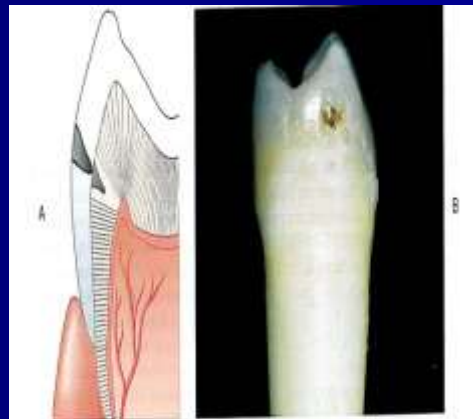
***1. Position of tooth***

***2. Morphology of tooth***

***3. Structure of teeth***

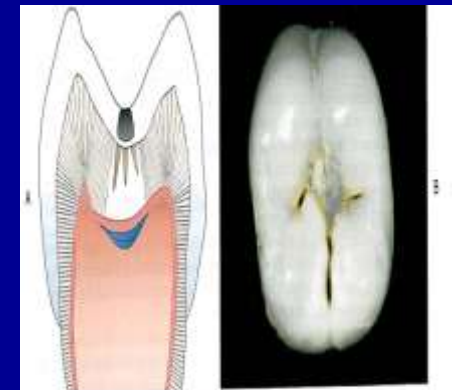
***4. Fluorides***

***5. Genetic factors***



**FIGURE 3-2**

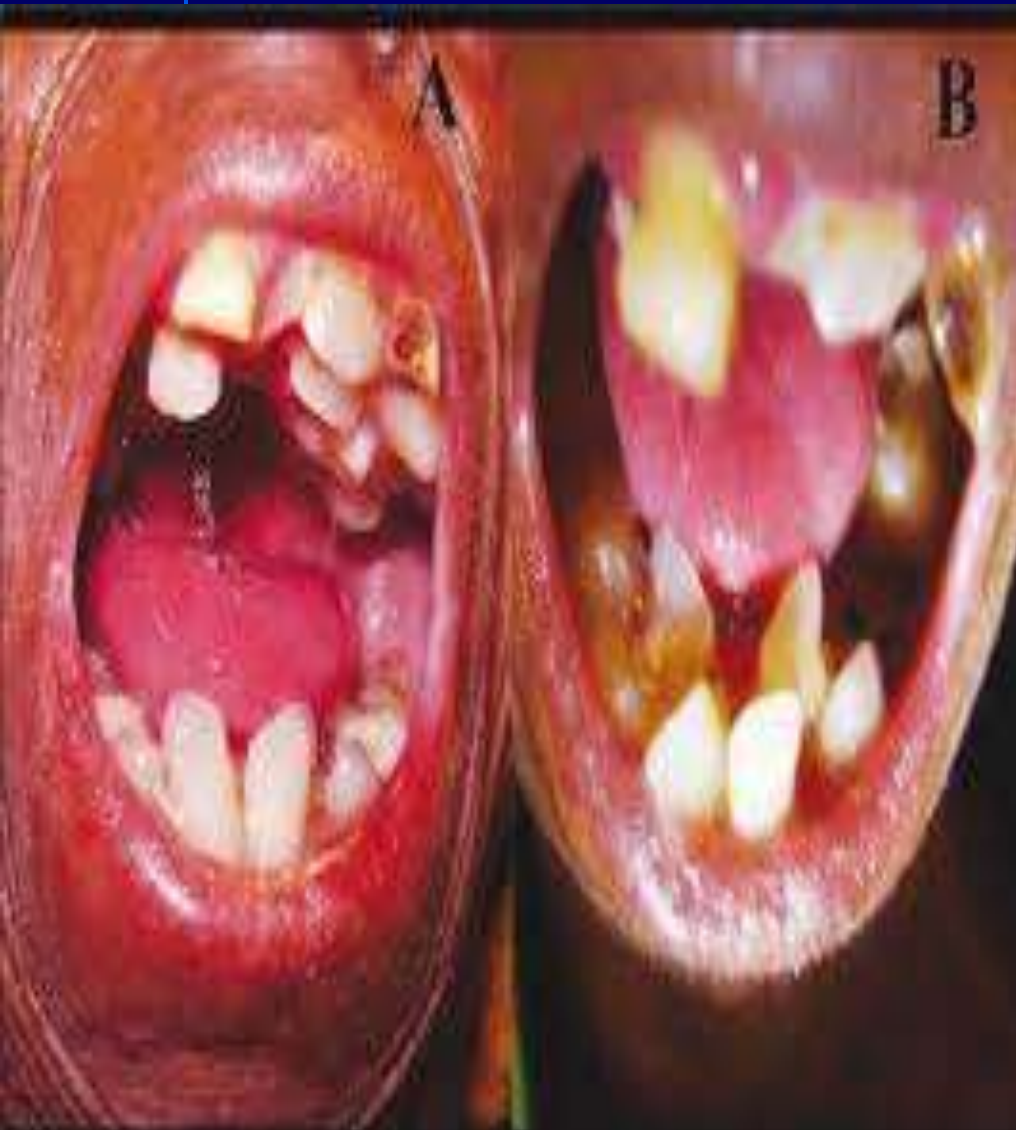
**Smooth-surface caries.** A, The characteristic triangular shape of these lesions that corresponds to the orientation of the enamel rods and the dentinal tubules. B, Clinical appearance of early enamel caries on the smooth interproximal surface, exhibiting the central brown zone of disintegration.



**FIGURE 3-1**

**Pit and fissure caries.** A, The characteristic shape of lesions demonstrating a small triangle-shaped lesion in the fissure of the occlusal enamel (gray block) that appears narrow at the surface but wide at the dentoenamel junction to provide an even greater involvement of dentin (dark brown). The pulp of the tooth reacts with the deposition of reparative dentin (blue). B, Clinical appearance of molar with fissure caries, exhibiting the black area of disintegration at the base of the fissure and the demineralized and undermined white opaque area surrounding the enamel.

1. Upper-Posterior teeth
2. Crowded teeth





# 2. Morphology of tooth

**1. Pits and fissures** increase susceptibility to caries because of creation of stagnation areas.

2. The position and shape of contact areas also influence caries involving **proximal surfaces**.

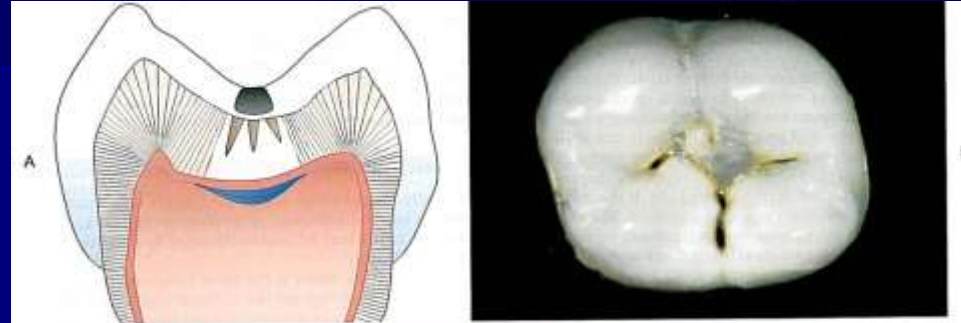


FIGURE 3-1

**Pit and fissure caries.** A, The characteristic shape of lesions demonstrating a small triangle-shaped lesion in the fissure of the occlusal enamel (gray-black) that appears narrow at the surface but wider at the dentoenamel junction to provide an even greater involvement of dentin (dark brown). The pulp of the tooth reacts with the deposition of reparative dentin (blue). B, Clinical appearance of molar with fissure caries, exhibiting the black areas of disintegration at the base of the fissure and the demineralized and undermined white opaque areas surrounding the enamel.

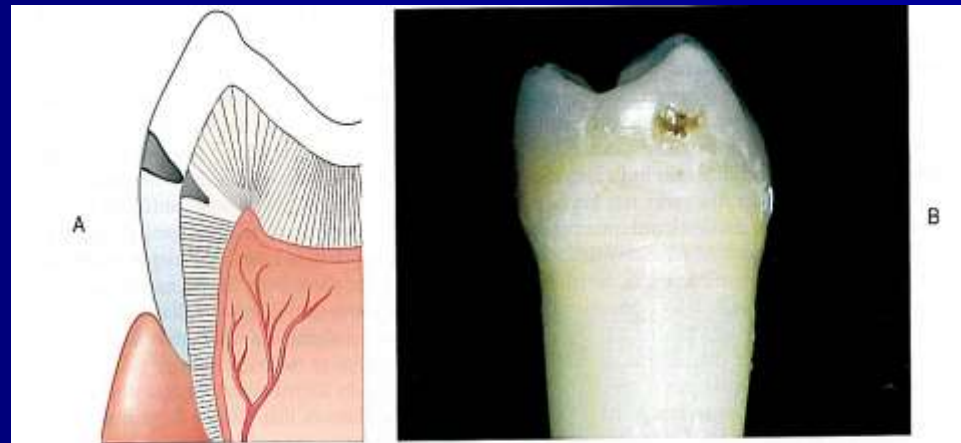


FIGURE 3-2

**Smooth-surface caries.** A, The characteristic triangular shape of these lesions that corresponds to the orientation of the enamel rods and the dentinal tubules. B, Clinical appearance of early enamel caries on the smooth interproximal surface, exhibiting the central brown zone of disintegration.

### ***3. Fluorides***

1. Fluoride is a naturally occurring constituent of our food and drinking water.
2. It is present either as bound fluoride or in ionic form in solution.
3. Ingestion of fluoridated water in concentrations more than 1.2 PPM, at the time of tooth mineralization, is associated with a form of enamel.
4. **Hypoplasia** known as enamel fluorosis or mottling.  
The appearance of this hypoplastic enamel is due to the toxic effect of fluoride on the ameloblasts.  
Mottled enamel, though hypoplastic, is more resistant to dental caries.



## ***4. Structure of teeth***

Caries may progress faster in

**hypoplastic** and or

**hypocalcified** teeth if

compared to normal.

## ***5. Genetic factors***

1. Hereditary has a minor influence on resistance to caries.
2. There is low incidence of caries in certain families which may be due to inheritance of a more favorable tooth shape or anatomy .However, it is difficult to exclude the role of common dietary habits in related persons.





## 2. Carbohydrate



# Factors affecting cariogenicity of carbohydrates

**a. Type of carbohydrates** : monosaccharide give more caries as they are easily break down, diffuse rapidly into plaque and they are most frequently used.

**b. Total amount of carbohydrates intake**: Increased Intake, increases caries activity.

**c. Frequency of carbohydrates intake** between meals increases caries activity.

**d. Consistency and texture of carbohydrates**; Sticky carbohydrates increases caries as they remain attached to the tooth.

## Role of Carbohydrates



Role of Carbohydrates

## Fermentable Carbohydrates





# 3. Micro-organisms

# 3. Micro-organisms

## *Types of bacteria involved in caries*

### 1. Acidogenic bacteria

#### a. Lactobacilli

#### b. Streptococcus mutans.

### 2. Proteolytic bacteria

### 3. Chromogenic bacteria.

#### Role of microorganisms

- *S. mutans* : development of early carious lesions in enamel
- Lactobacilli : associated with dentinal caries
- Actinomyces : associated with root surface caries
- Vellionella: possibly anti-cariogenic



## ***Essential features of cariogenic bacteria***

1. Formation of acids (acidogenic) i.e. PH is less than 5.5.
2. Can live and grow in acid (acid uric).
3. Production of abundant polysaccharides for formation of plaque matrix.
4. Production of insoluble polysaccharides that resist dissolution by dietary or salivary flow.
5. Have attachment mechanisms for firm adhesion to tooth surface.

# Plaque



A thin, sticky film that builds up on teeth and contributes to tooth decay.



# What is Dental Plaque

*Dental plaque is a biofilm which builds up on and surrounding the teeth. Plaque is Sticky and usually colorless. It forms every day on teeth.*



For more details visit  
<http://www.smilecareworld.com>

smile care

EXPERT DENTAL CENTRE

## Plaque and Your Teeth

- Plaque is the sticky, colorless film of bacteria that forms on teeth. It makes teeth "feel fuzzy" to the tongue and is most noticeable when teeth are not brushed.
- It is made up of invisible masses of harmful germs that live in the mouth and stick to the teeth.



- Some types of plaque cause tooth decay
- Other types of plaque cause gum disease

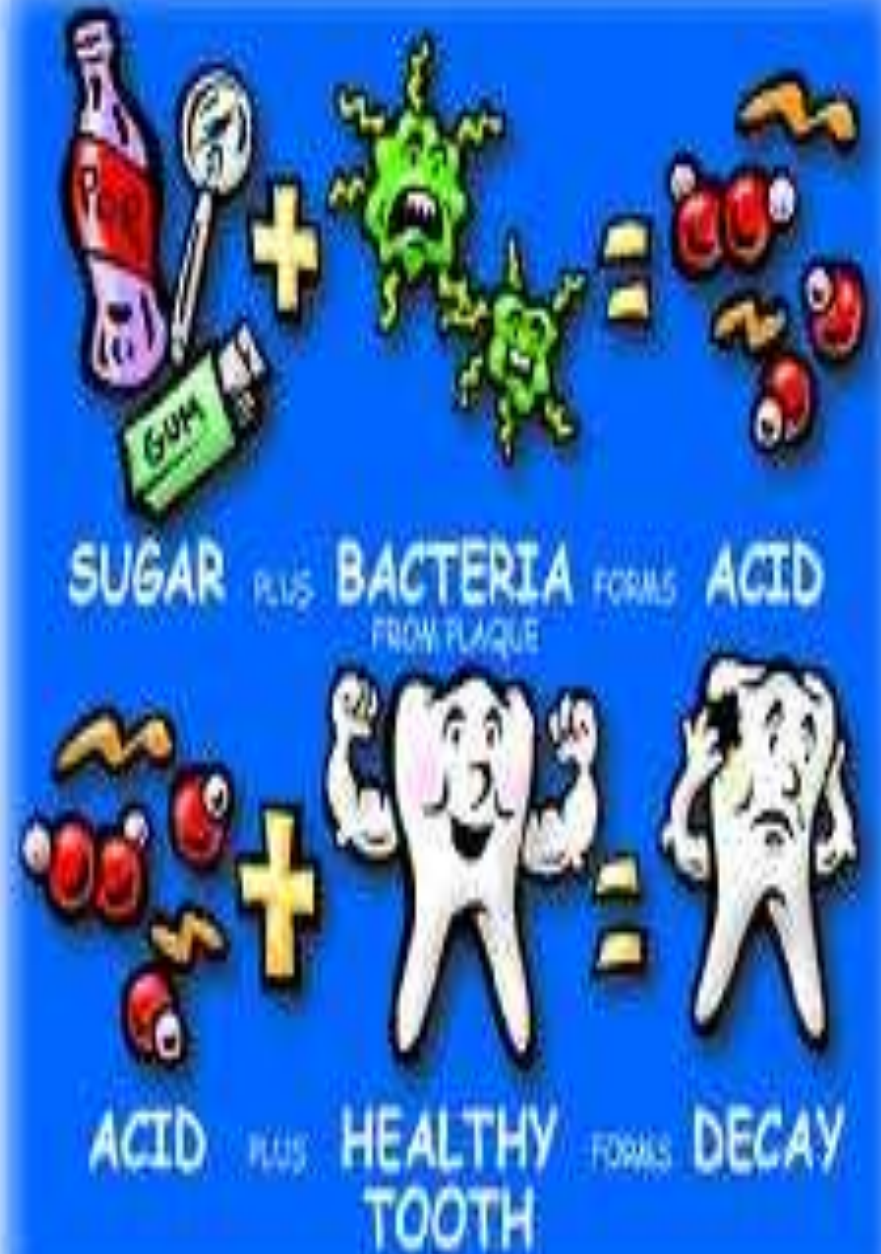




# What Causes Plaque and Why Is It Harmful?



- Plaque develops when foods containing carbohydrates (sugars and starches) such as milk, soft drinks, raisins, cakes, or candy are frequently left on the teeth. Bacteria that live in the mouth thrive on these foods, producing acids as a result. Over a period of time, these acids destroy tooth enamel, resulting in tooth decay.
- It can also develop on the tooth roots under the gum and cause breakdown of the bone supporting the tooth



# ***Role of dental plaque***

1. Plaque is important in the etiology of caries because acid is generated within its substance.
2. Dietary sugars diffuse rapidly within plaque where they are converted to acids by bacterial metabolism.
3. The pH of the plaque falls as much as 2 units within 10 minutes after the ingestion of sugar.

At a critical pH, about 5.5, mineral ions are liberated from the hydroxyapatite crystals of the surface enamel and diffuse into the plaque.

# ***Composition of dental plaque:***

***1-Micro-organisms:*** e.g. Streptococcus 95%, filamentous, lactobacillus, Etc.

***2- Matrix:*** formed

1. Protein: from saliva, bacteria & crevicular fluid.
1. Carbohydrates : polysaccharides formed by bacteria
3. Lipids: bacterial in origin.

***3. Inorganic components:*** Ca, phosphate, potassium Mg, Na, fluoride.

***4. Water:*** plaque contains about 80% water and 20% solids.

*pH of plaque is 7.1 in caries free persons*

*but 5.5 in persons with extreme caries.*



# ***Factors affecting cariogenecity of the plaque***

## ***1. Thickness***

Plaque cannot produce caries unless it acquires a **critical thickness**.

## ***2. Bacterial population of the plaque***

**Type of bacteria** will determine its cariogenic effect and whether the formed plaque will be involved in periodontal disease or in caries process.

# How can plaque formation be prevented

## Summary



- Brush at least twice daily
- Floss at least once a day
- Use water or tooth past which contain fluorides
- Visit your dentist regularly



# Dental pellicle

Dental pellicle (salivary glycoprotein) is deposited, and then colonization of bacterial flora takes place.



# Extrinsic factors

***1. Saliva***

***2. Diet***

# ***1.Saliva***

- a. Composition:** caries free persons have greater amount of **ammonia**.
- b. pH:** **normal** salivary pH is alkaline, low neutral or slightly pH increase the process of demineralization of enamel.
- c. Quantity:** **Xerostomia** leads to rampant caries.
- d. Viscosity:** High caries index is associated with **thick, mucinous** saliva.

## **Increase of caries incidence**

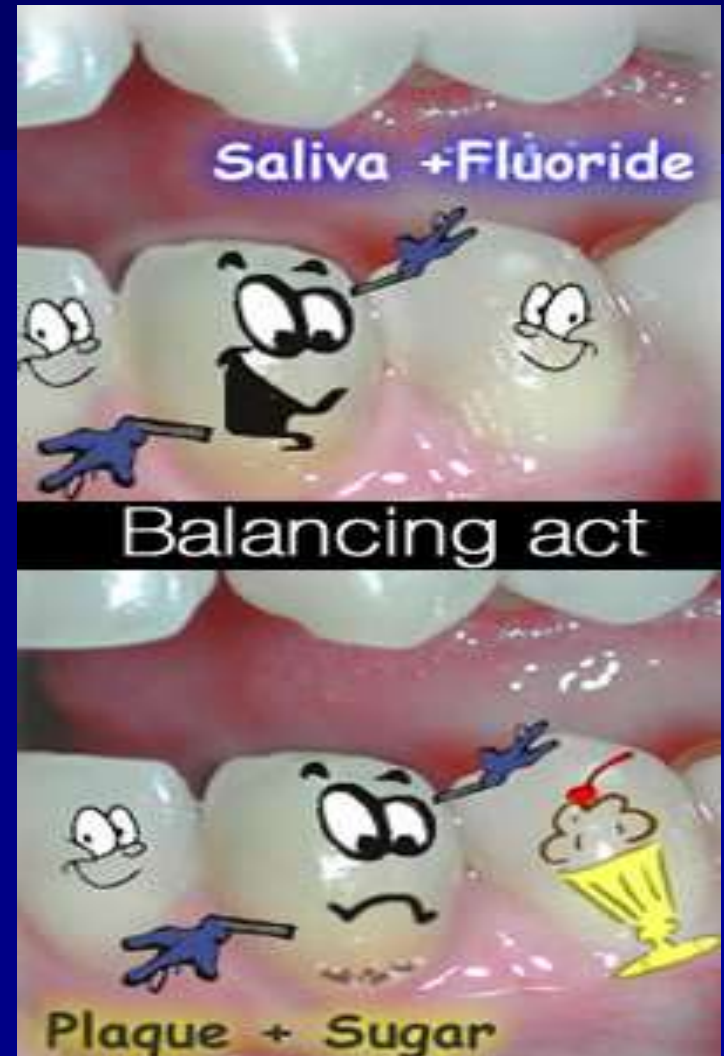
1. No ammonia in composition in saliva.
2. Decrease in amount of saliva.
3. Low of pH.
4. Thick, mucinous saliva.

# ***Role of saliva in caries production***

1. Saliva is the medium in which plaque develops & works

***Its role can be summarized in the following points:***

- a. Formation** of pellicle from the salivary glycoprotein.
- b. cleaning effect:** (Xerostomia increases caries incidence)
- c. Buffering effect:** Depends on its bicarbonates & phosphate.
- d. Saliva has antibodies:** (IgA) that kills microorganisms.
- e. Other antibacterial** substance like lysozyme, peroxidase & lactoferrin.





## ***2. Diet***

**a- Physical factor:** Unrefined foods are less cariogenic than modern soft foods.

**b- Local factor:**

**1. Carbohydrate content:** Food rich in Carbohydrate is more cariogenic than other types of food.

**2. Calcium & phosphate intake:** They may reduce caries index.

**3. Fluoride content:** Fluoridation of drinking water reduces caries incidence as it forms fluoroapatite crystal which is more resistant to caries than hydroxyapatite.

**4. Vitamin content:** Vitamin D, K and B6 may reduce caries incidence.

# Vitamin D



The book, available at [www.lambert-publishing.com](http://www.lambert-publishing.com), is a valuable resource for health professionals and the general public alike. It provides a comprehensive overview of the importance of nutrition in maintaining good health, and includes a wealth of information on the latest research in the field. The book is written in a clear, concise, and easy-to-understand style, making it an ideal resource for anyone interested in improving their diet and overall health.



Book and Food and Drink  
**The Essentials of Good Nutrition**  
 Barbara Fennell and Dr. John M. Fennell

Book and Food and Drink

Available in paperback and hardcover. The book is written by a team of experts in the field of nutrition, and provides a comprehensive overview of the importance of nutrition in maintaining good health. It includes a wealth of information on the latest research in the field, and is written in a clear, concise, and easy-to-understand style.



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Book and Food and Drink



Water fluoridation



Proper diet



Soft food





# Theories of dental caries



# Etiology of dental caries

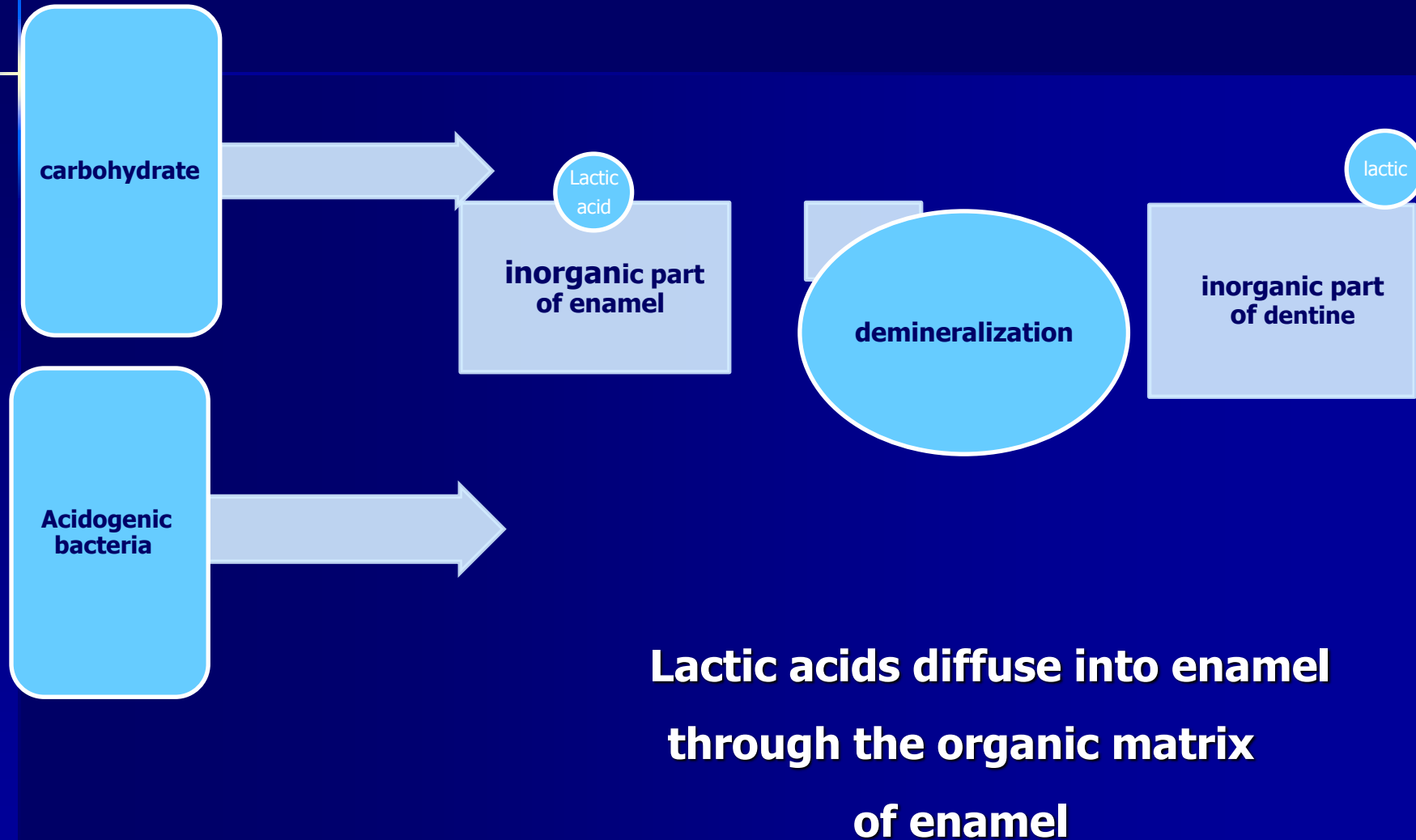
Various theories for the etiology of dental caries have been proposed:

*I- Acidogenic theory (Miller's theory)  
(chemico- parasitic theory)*

*II- The proteolytic theory*

*III- The proteolysis – Chelation theory*

# Acidogenic theory (Miller chemicoparasitic theory)



# Proteolytic bacteria

**Proteolytic •  
bacteria**

**organic •  
matrix of  
dentine**

**digestion •  
and  
dissolution**



# Role of acidogenic bacteria

## ***1. Streptococcus mutants***

- These acidogenic bacteria fulfill all the characteristics. It is called mutants as it undergoes mutation from round to rod shape in different PH.

- It has the ability to polymerize sucrose to give high molecular weight polysaccharide (dextran) which helps plaque to attach to the tooth.

**N.B** *Dextran is sticky, gelatinous substance that enables the microorganisms to adhere to the tooth and form the plaque that initiates caries in smooth surface.*

***2. Other streptococci e.g. strep. Sanguis, slaveries & mitis*** produce pit & fissure caries in gnotobiotic rats (germ-free rats).

***3. Lactobacilli*** play a role in root surface caries & not in enamel Caries.

# Role of acid

carbohydrates

bacteria

enzymatic breakdown



**Acid formed**

(Lactic acid & Butyric acid)

dental  
plaque



Acid should be localized to  
tooth surface for long period.

pH at 'plaque-tooth interface' less than 5.5 → enamel demineralization process begins

loss of calcium surface and



bacterial plaque is not  
regularly removed from the  
tooth surface.

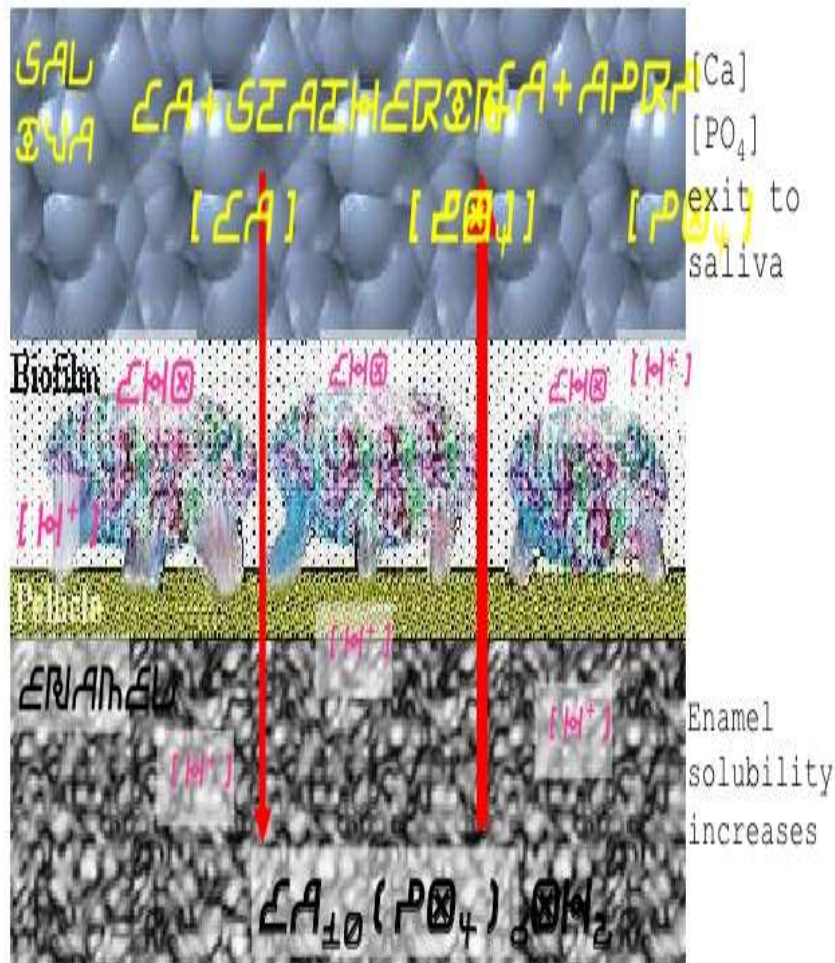
## Enamel caries re-mineralization (*Caries reversibility and consolidation*)

1. Early white enamel caries could be arrested by **topical application of fluoride in children**
2. Fluoride ion plays a role in stimulating remineralization by increasing the rate of calcium deposition.
3. **Calcium phosphate solution** may also cause hardening of E, if cavitations have not occurred.
4. Remineralization of E occurs by **precipitation of mineral salts from saliva**



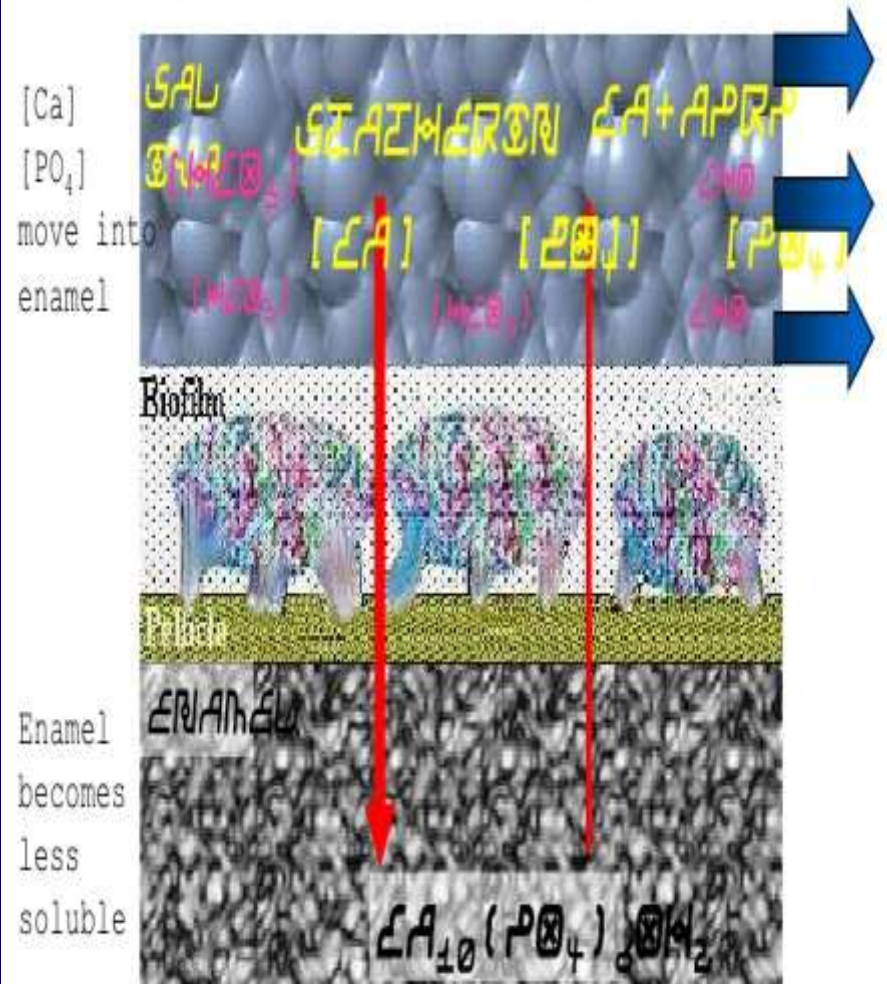
# Demineralization

Dietary CHO + biofilm = lactic acid; diffusion into enamel = local pH drop



# Remineralization

Saliva flow clears CHO; salivary  $HCO_3^-$  returns pH to normal



demineralization process is reversible  
provided that the  
acidogenic properties of the biofilm are  
neutralized.



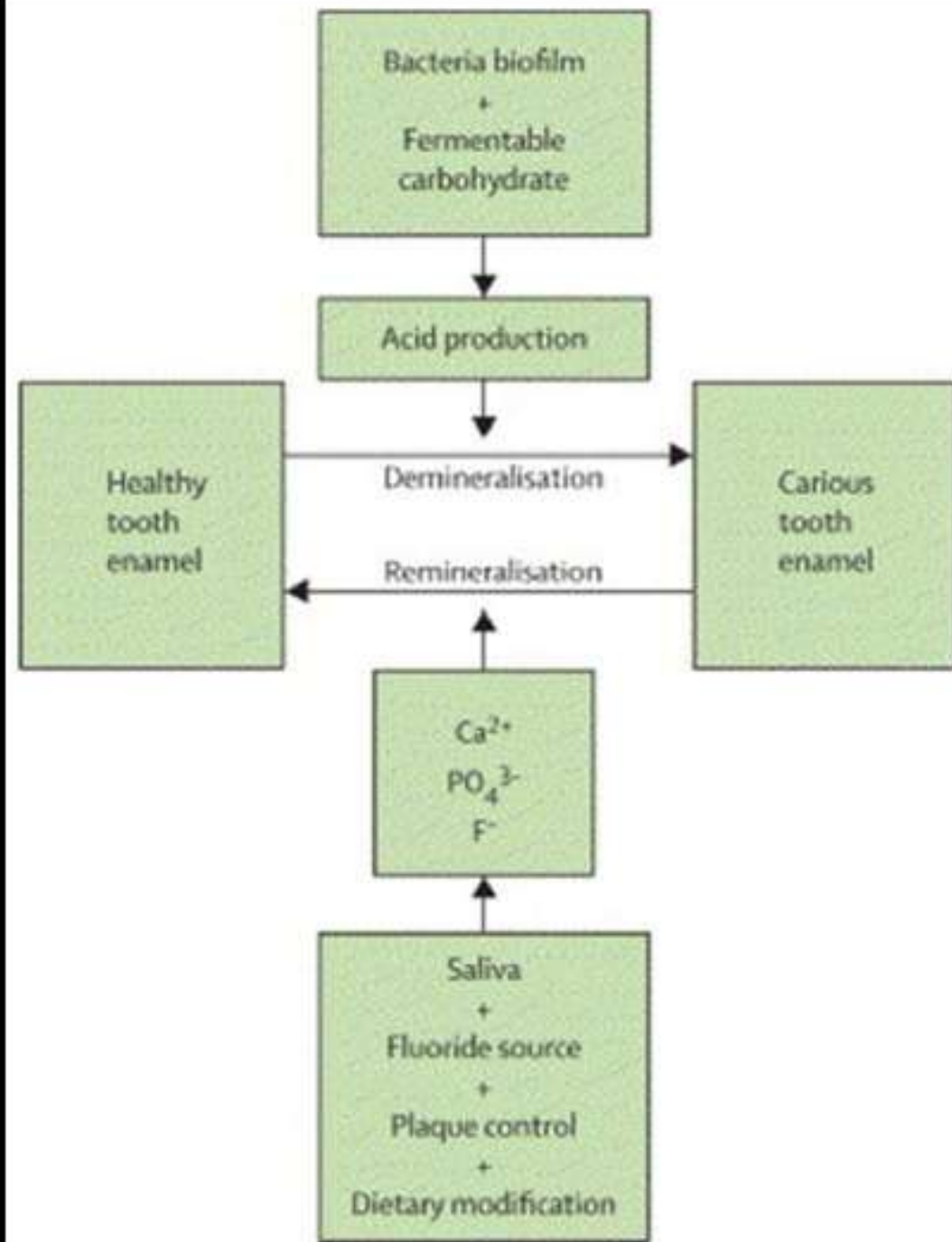
Buffering capacity of saliva

If dietary carbohydrates are removed / pH  
= 7 → REMINERALISATION occurs

Once the pH returns to higher than the  
critical point, demineralization is  
arrested and minerals can be added back to  
the partially dissolved enamel  
crystallites.

# Pathogenesis of Dental Caries

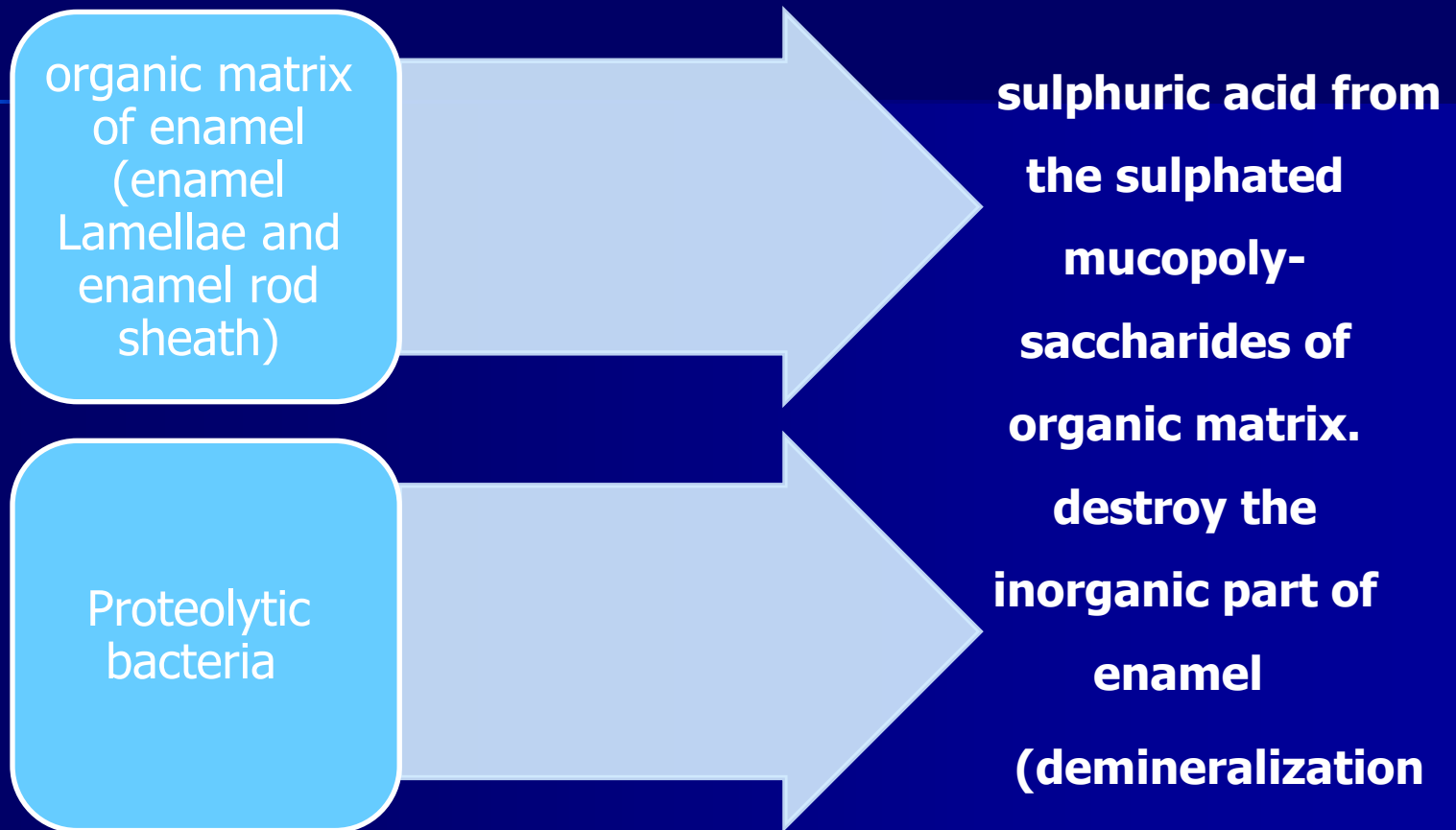
- ✓ Fermentation of dietary sugars by Oral micro-organisms
- ✓ De-mineralisation
- ✓ Re-mineralisation
- ✓ Further demineralisation and Cavitation
- ✓ Initiation / Formation of Caries





# **Proteolytic theory**

# The proteolytic theory



Bacteria may degrade protein in absence of carbohydrates  
.e.g. in deep dental caries.

# Caries, a Proteolytic process

Proteolytic enzymes liberated by cariogenic bacteria



destruction of the organic matrix



detachment of inorganic crystals from one another



collapse of whole structure

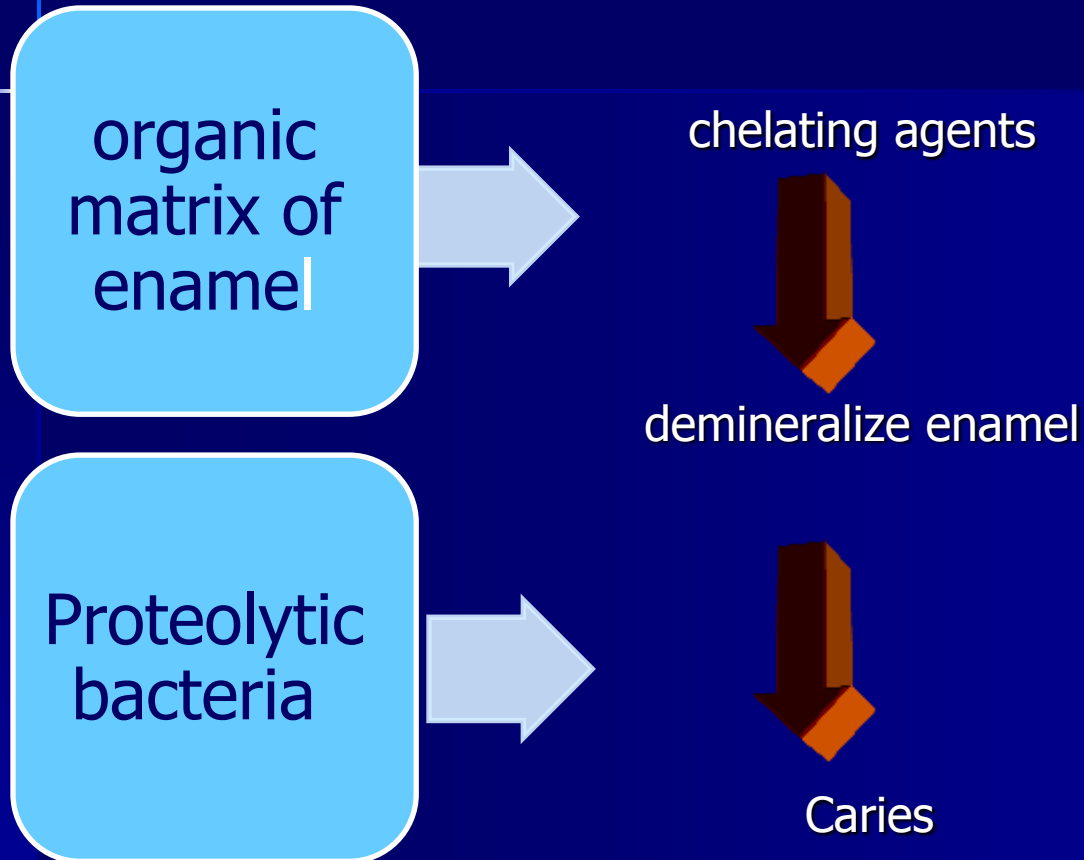


CAVITATION.

# **PROTEOLYSIS CHELATION THEORY**

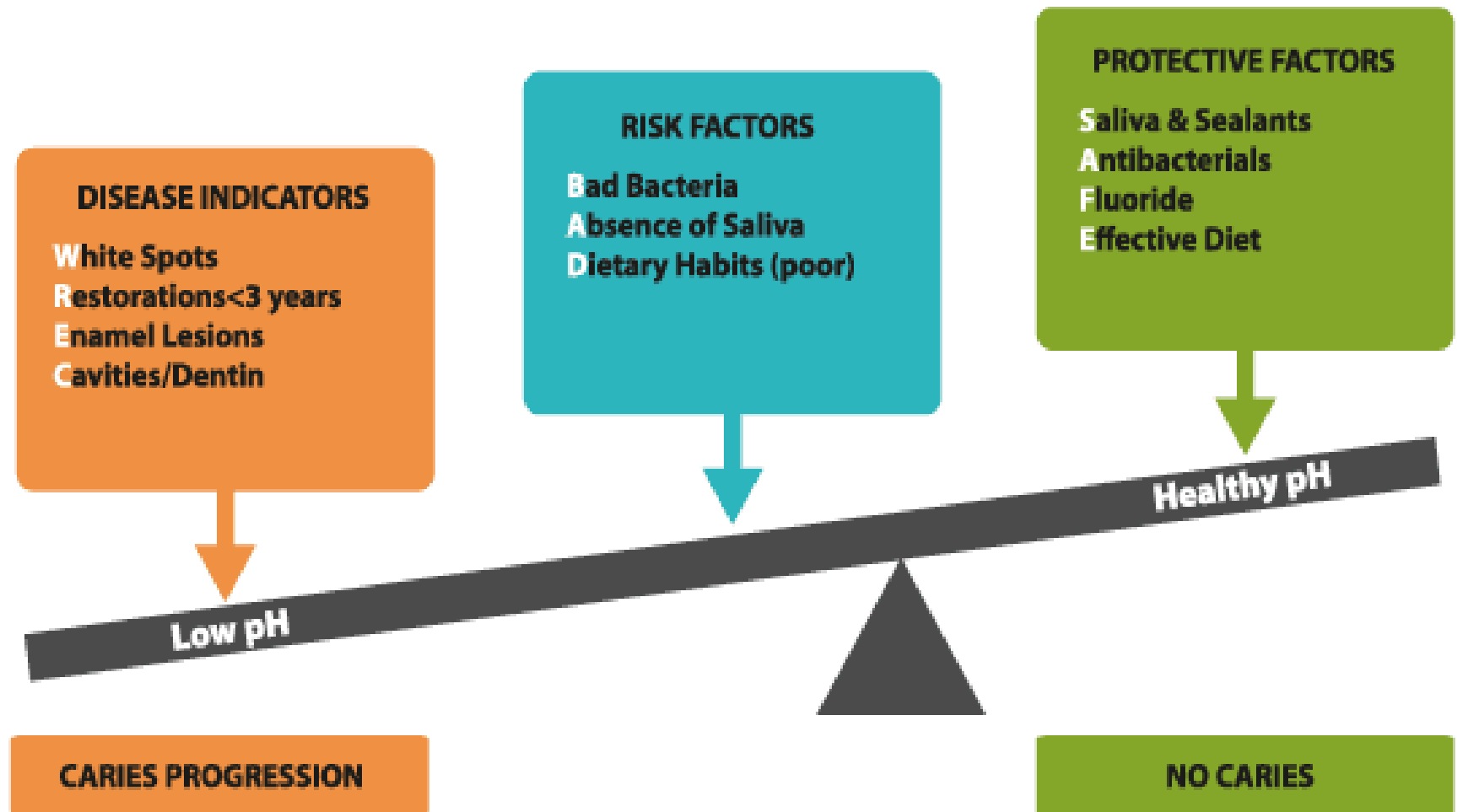


# The proteolysis – Chelation theory



Chelation is the ability of certain organic compounds to associate with other ions or atoms by covalent bonds.

# The Caries Imbalance



**Figure 8. The Caries Imbalance – John B. Featherstone, MSc, PhD**

J. B. Featherstone, S. Domejean, L. Jenson, M. Wolff, and D. Young, "Caries Risk Assessment in Practice for Age 6 Through Adult," *CDA Journal* 35, no. 10 (October 2007).

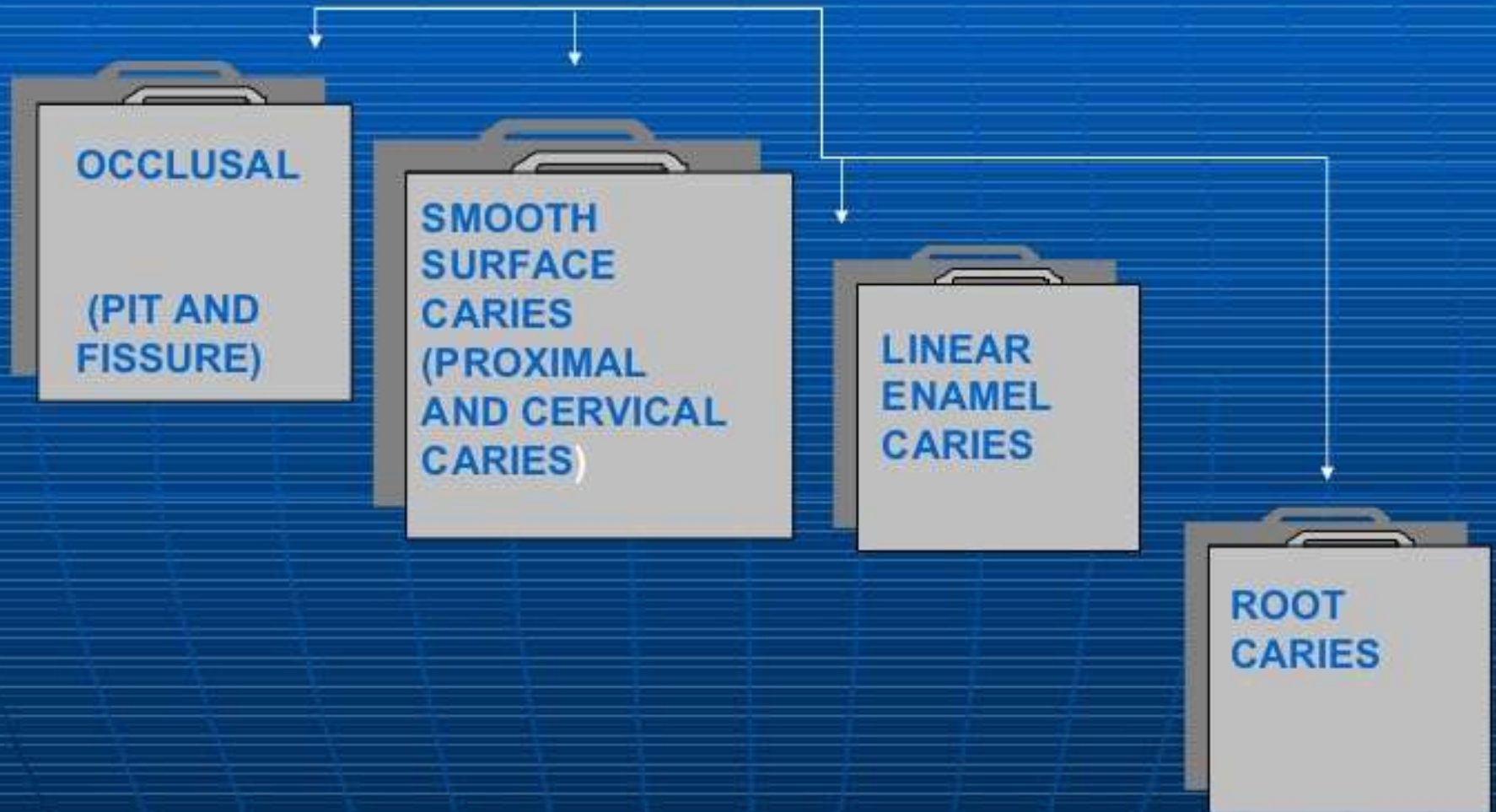
# Classification of dental caries

A night sky photograph featuring the Milky Way galaxy stretching across the upper half of the frame. In the foreground, a large, light-colored rock formation with vertical fissures is visible on the left, and dark silhouettes of other rock formations are on the right. The overall scene is a composite of a celestial and a terrestrial landscape.

Capital Reef National Park  
(c) Wally Pacholka



# 1. BASED ON ANATOMICAL SITE



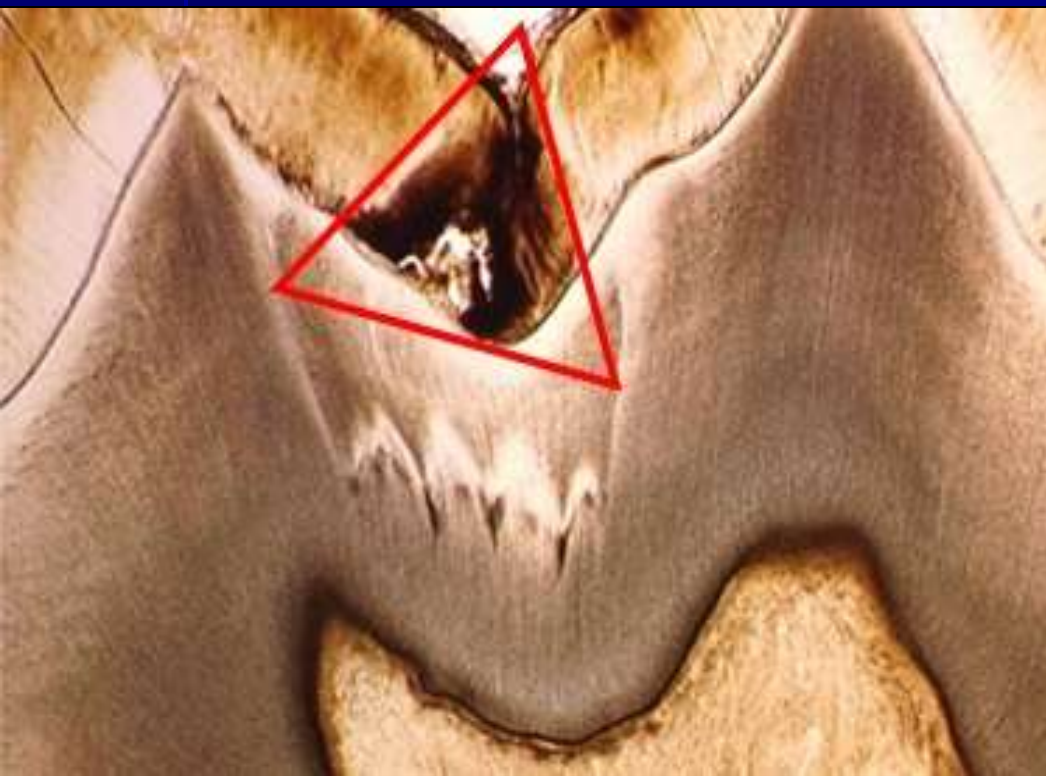
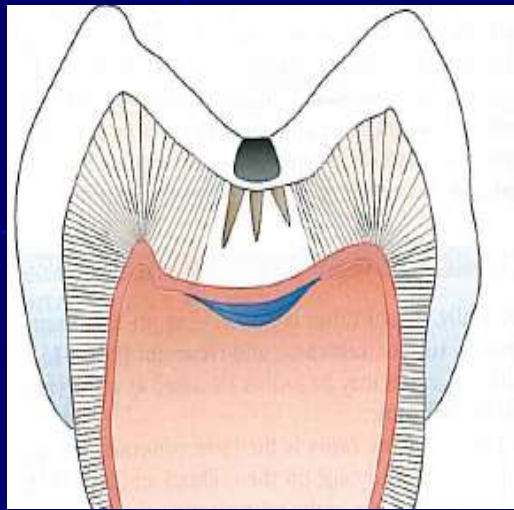


# Classification of dental caries according site of attack

## *Pit and fissure caries*

1. It is the most common type of dental caries.
2. It appears on the occlusal surface of molars and premolars, on the buccal and lingual surfaces of molars, and lingual surface of maxillary incisors.
3. It is the most destructive because it quickly goes deeply into the dentine.
4. **Early caries** detected by a brown discoloration of a fissure in which probe 'sticks'. The staining is due to exogenous pigmentation of E from food, tobacco & bacteria.
5. The enamel bordering the pit or fissure appears opaque "bluish-white" as it becomes undermined by caries, which may fracture suddenly under stress of mastication .
6. Early fissure caries is more difficult to diagnose by probing. Enamel (E) which appears clinically sound can overlay extensive caries of the dentine (D). This is due to the lateral spread caries at the amelodentinal (ADJ) junction.

# Pit & fissure caries

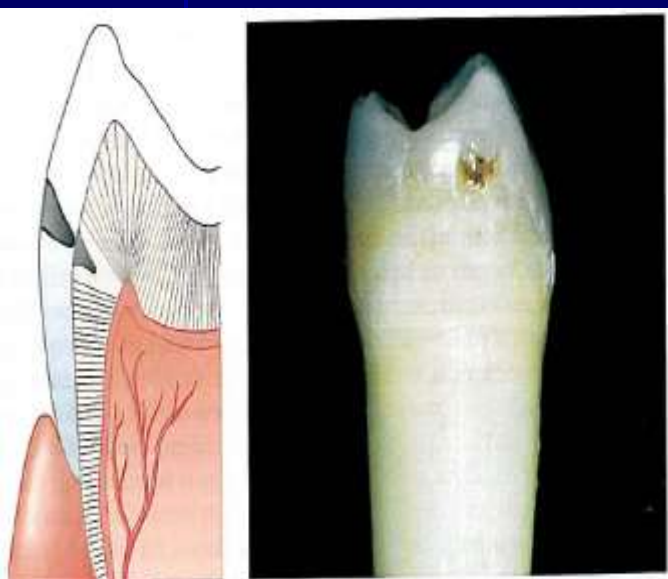


## ***2-Smooth surface caries***

- 1.It is the less common type of caries.
  - 2.It occurs on the interproximal areas of the teeth that are not self-cleansing.
  - 3.On occasion the cervical regions of the buccal and lingual surfaces of the teeth will become involved.
  4. A proximal caries begins just below the contact point as well **demarcated chalky-white opacity of the enamel**. At this stage there is no loss of continuity of the enamel surface and the lesion cannot be detected by a probe or on routine radiographs.
  - 5.The white spot lesion may become pigmented yellow or brown and may extend buccally and lingually.
  - 6.As the caries progresses, the surrounding enamel and dentine becomes bluish-white.
  - 7.The surface of the lesion becomes roughened before frank cavitation occurs. There are no consistent radiographic features which enable identification of enamel lesions that have been cavitated from lesions where the surface is still intact.
- In most cases, a radiolucency that dose not extends to the ADJ dose not represent a frank cavity.



# Smooth surface caries



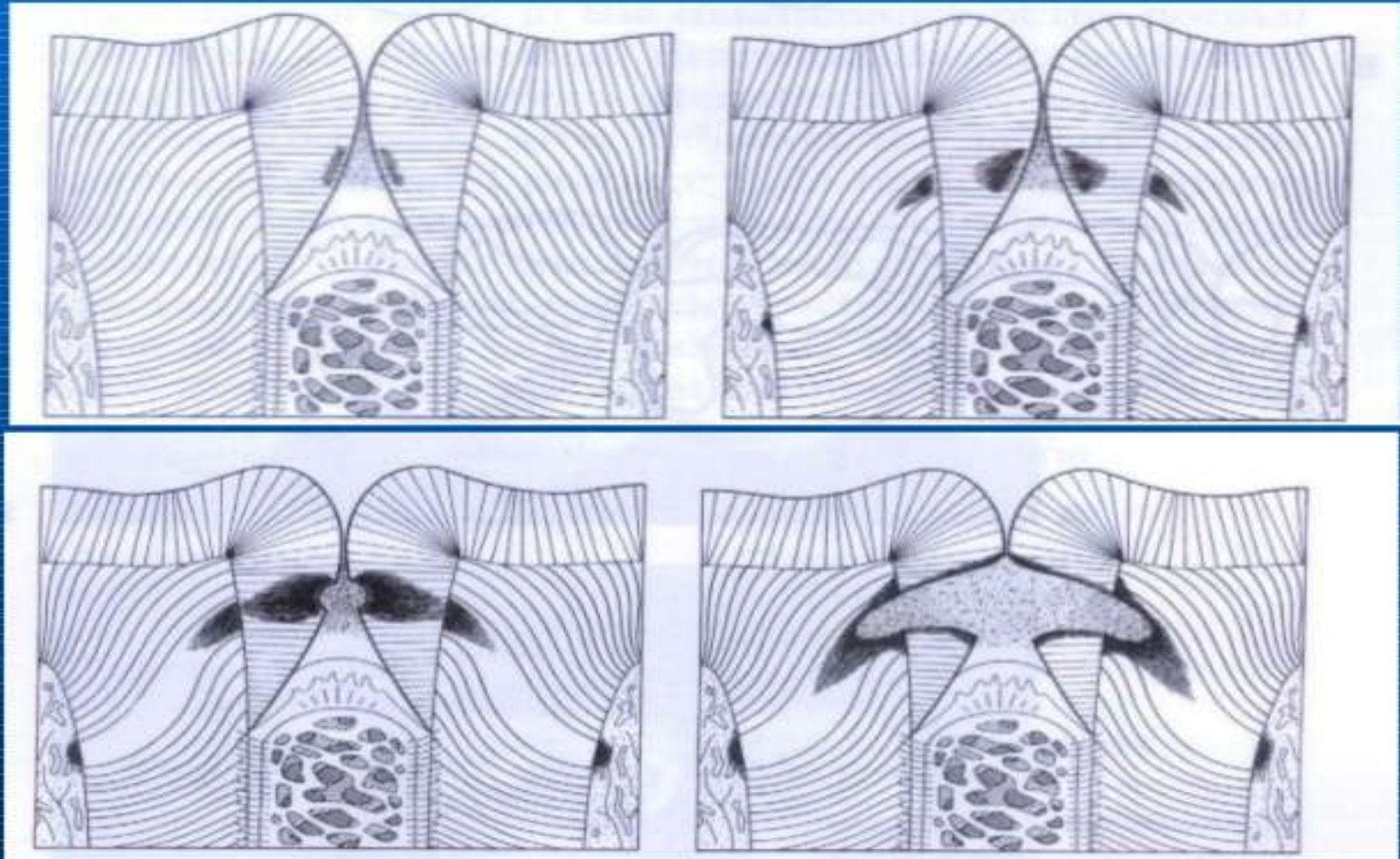
## Cervical caries





# Classification of dental caries according site of attack

## *Smooth surface caries*



# proximal caries, early enamel lesion





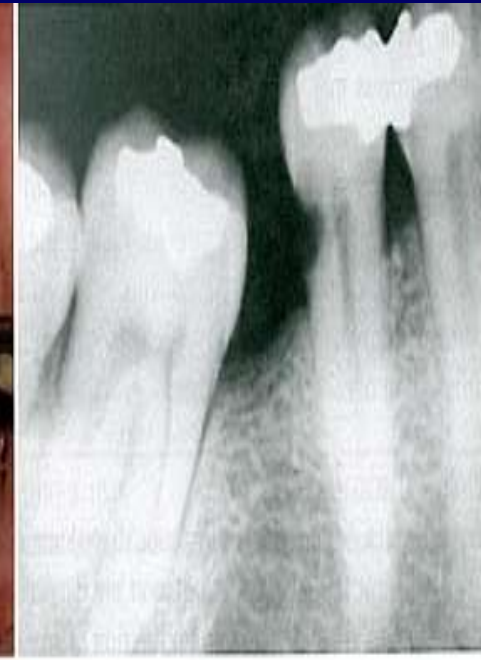
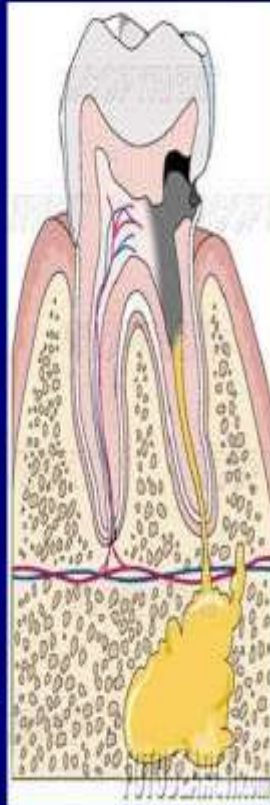
# ***Cemental (root) caries***

This occurs when the root is exposed to the oral environment as a result of periodontal disease. It is saucer shaped (concave surface contour) with ill defined boundaries

Root Caries

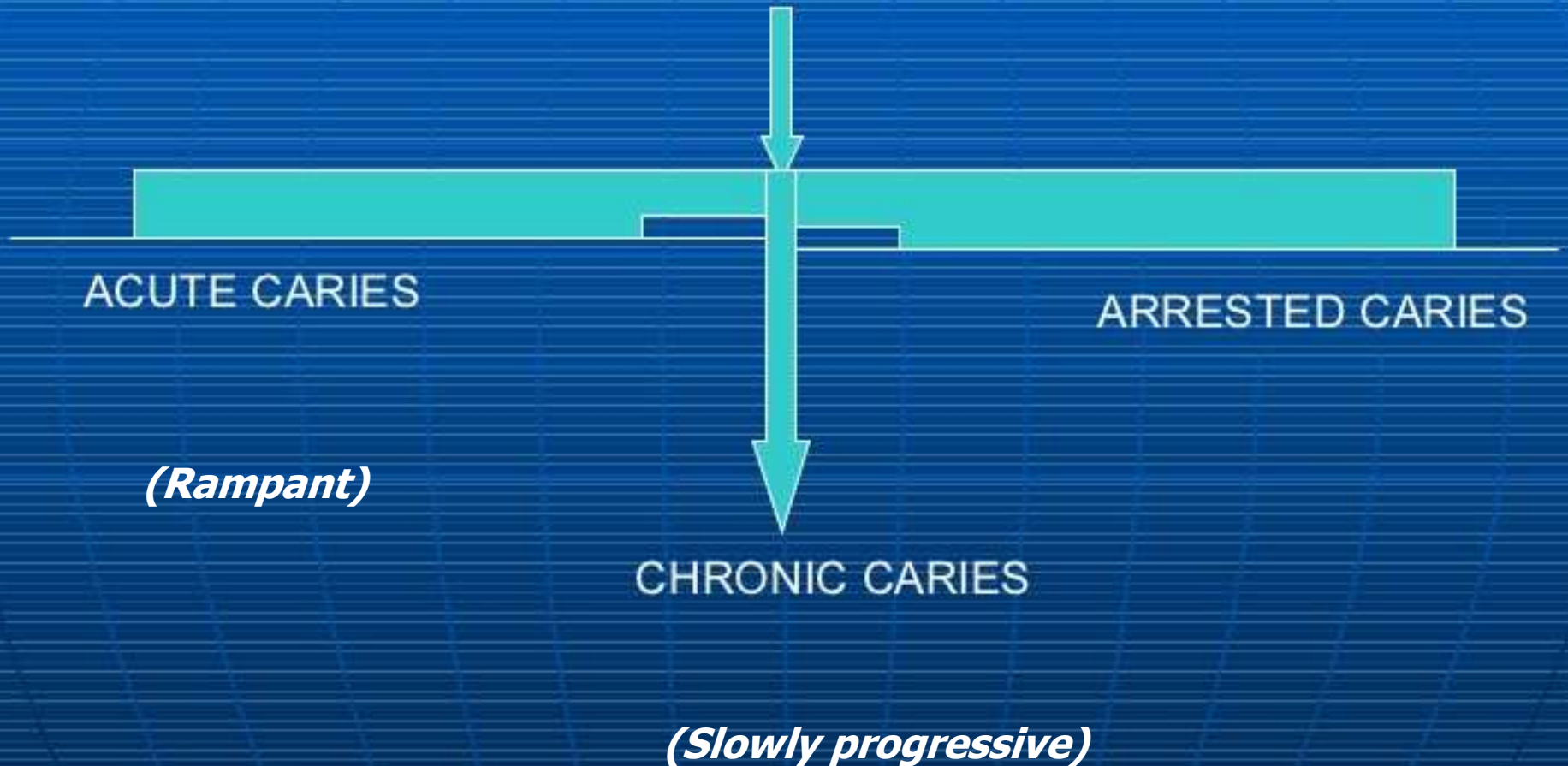


# Root or cemental caries





## 2.BASED ON PROGRESSION



# ***1.Acute caries (Rampant)***

- 1.This is rapidly progressing caries involving many or all of the erupted teeth often on surfaces normally immune to caries.
2. It leads to early involvement of the pulp (**pulp exposure & sensitive teeth**).
3. It is rampant caries affecting deciduous teeth in babies due to prolonged use of milk after eruption of deciduous teeth called (***Nursing bottle caries***).
4. The maxillary incisors are destructed followed by molars if the habit is prolonged.



***Nursing bottle caries***

**Nursing bottle caries**



# Rampant caries





# Rampant caries



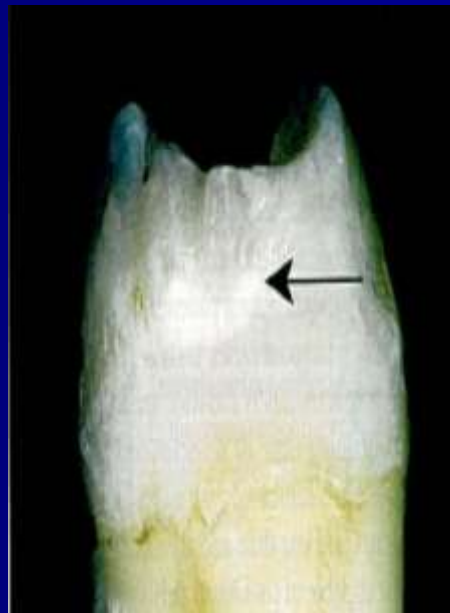


## ***Chronic caries (Slowly progressive)***

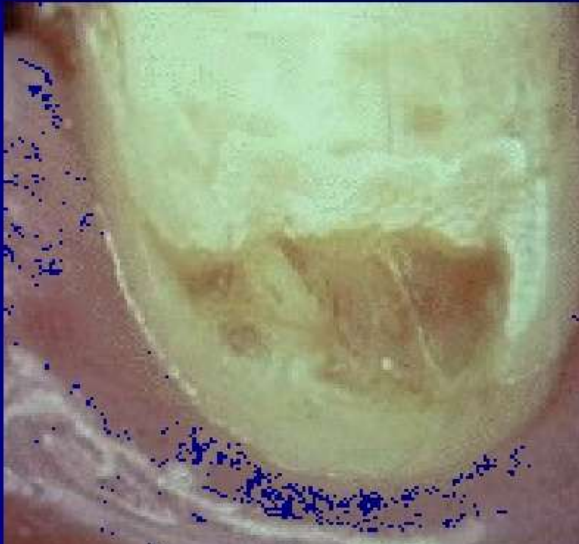
1. This is caries that **progresses slowly** and involves the pulp much later than in acute caries.
2. It is **most common in adults & No pain**
3. The slow progress allows time for defense reactions of the pulp and dentine (sclerosis and secondary dentine formation).

# 3-Arrested caries

1. Caries which becomes static & does not penetrate.
2. Sclerosing and secondary dentin is formed lead to arrest caries.
3. Usually occurs in occlusal surface with large open cavity, lack of food retention which leads to brown hard polished surface.
4. It occurs in proximal surface if adjacent tooth is extracted or following topical application of stannous fluoride.



# Arrested caries



- Exclusively seen in caries of **occlusal surface** with large open cavity in which there is lack of food retention
- Also on the proximal surfaces of tooth in cases in which the **adjacent** approximating tooth has been **extracted**



## Arrested caries

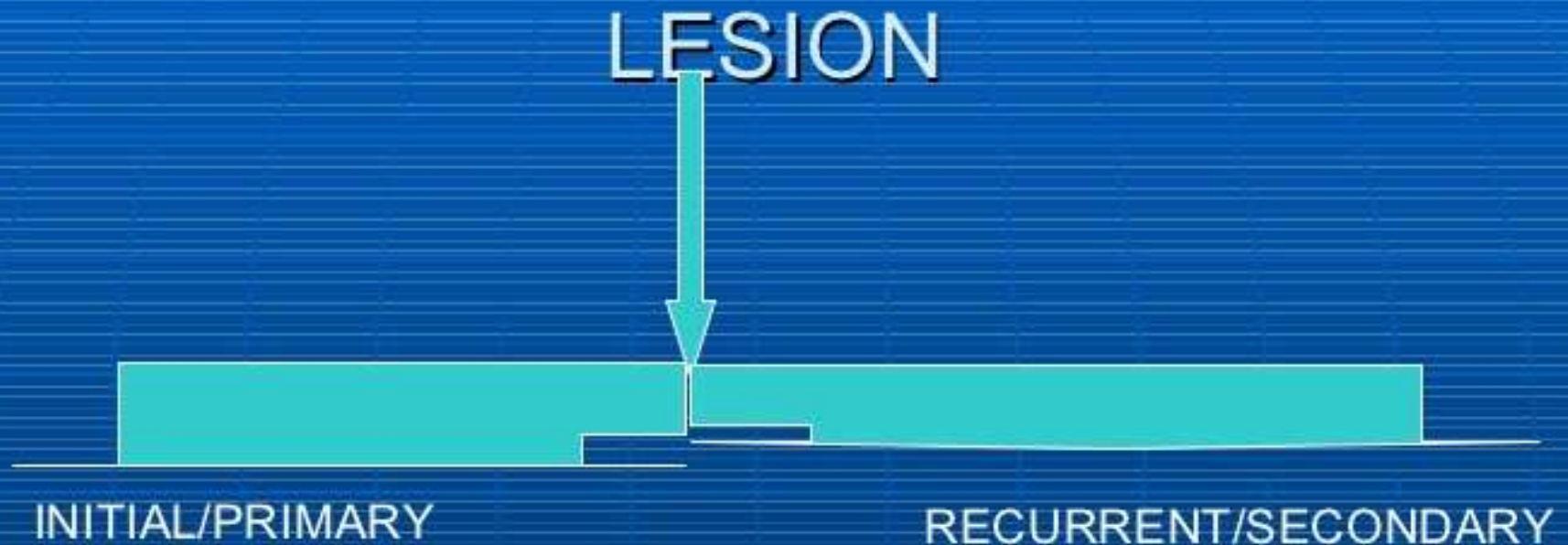
- No t  
prog
- Excl
- Larg  
supe  
deca  
burn  
hard



surface  
the  
shed

### III-Classification according to onset of occurrence

## 3.BASED ON VIRGINITY OF



**Caries occurs for first time**

- : **Occurs under restoration** due to
  - A. inadequate extension leading to retention of debris
  - B. poor adaptation of filling (leaky margin)
  - C. if all carious dentine is not removed.



# caries







# Pathological changes

# **Pathology of enamel caries**

## ***Macroscopic picture (Clinical picture of enamel caries)***

1. At first the lesion appears as a white chalky area.
2. Then it becomes rough to the probe and staining will occur due to the action of some chromogenic bacteria.
3. Later on cavitations will occur.



# Pit and Fissure caries

Due to Poor self-cleansing/ developmental faults of tooth

Early lesion

Region of  
caries

Carious  
rod

Undermining occurs through lateral  
spread at DEJ

May penetrate into dentine through  
dental tubules



In pit & fissure caries the apex of the lesion is on the surface and the base on ADJ, this is due to the flaring of enamel rods laterally from the bottom of the pit



# proximal caries, early enamel lesion



In smooth surface enamel caries, the carious lesion is cone-shaped, with the base of the cone on the enamel surface and the apex pointing towards the amelo-dentinal junction (ADJ).

# ***Microscopic picture***

**Enamel caries occurs in 4 phases:**

1. Initial phase
2. Bacterial invasion
3. Destruction phase
4. Phase of secondary enamel caries

# I-Initial phase divided into four zones:

**Zone I:** **Translucent zone**, it is more porous.

- In this zone, enamel contains 1% by volume of pores.

(Normal enamel contains only 0.1 % pores by volume).

Clearing (mounting) medium fills the formed pores giving this zone a well demarcated translucency from normal enamel.

**Zone II:** **The dark zone**, it is due to further demineralization.

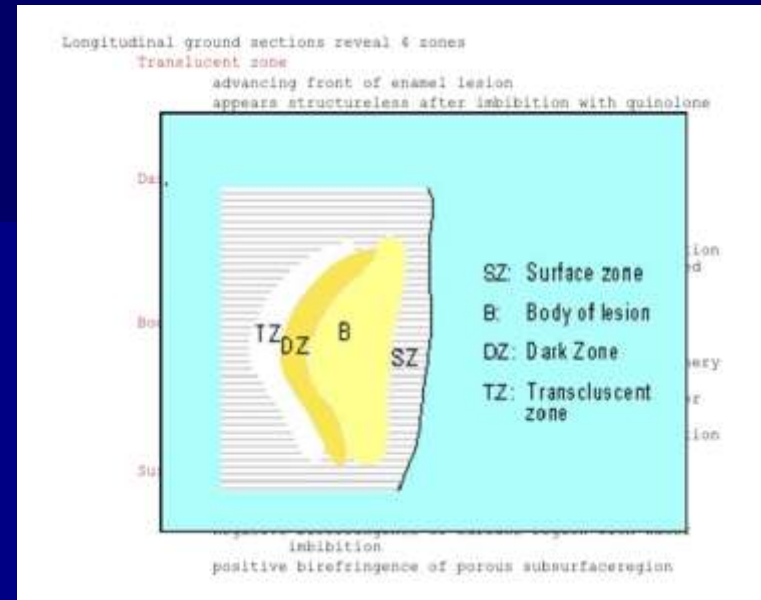
- It contains 2-4 % by volume pores.
- some of them are larger as a result of demineralization.
- Some pores are so small that mounting medium cannot enter and so filled by air and appear dark.

**Zone III:** **Body of the lesion**, it represents the largest area of the lesion.

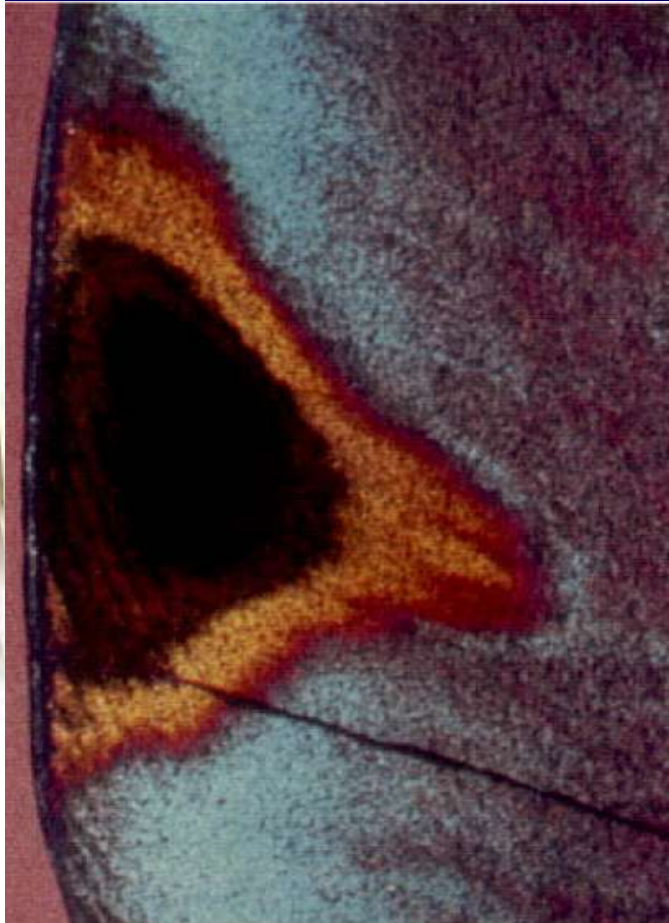
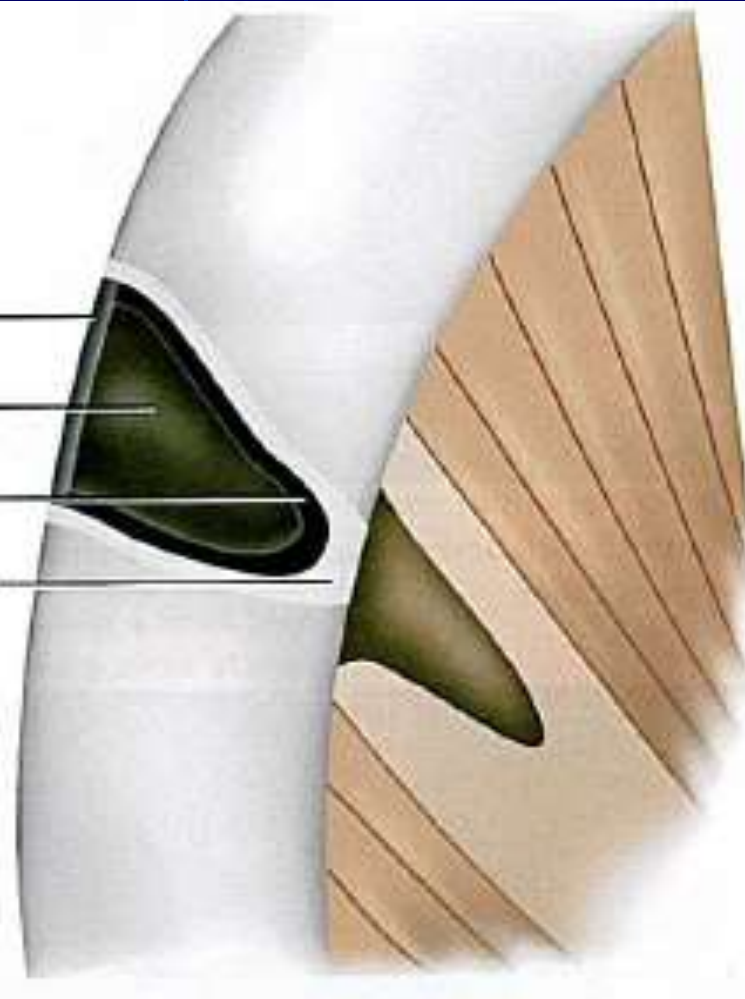
- Pores represent up to 25% by volume leading to area of great demineralization.

**Zone IV:** **The surface zone**, it is relatively unaffected as it has greater resistance due to presence of fluoride.

- This layer remains relatively normal. Cavitations occurs when this layer is lost.



# Early enamel caries





# II. Bacterial invasion

1. Bacteria invade enamel
2. Acids created sufficient pathways
3. cavitations occurs allowing bacteria to penetrate .

# III-Destruction phase

1. Loss of minerals
2. Proteolytic bacteria act on organic matrix of enamel
3. Complete destruction of the area.

## **IV-Phase of secondary enamel caries**

1. When acids and bacteria reach the ADJ they spread laterally along it.
2. It begin to invade enamel from beneath .
3. Enamel is undermined & affection of large number of dentinal tubules.





# Pathology of dentine caries



# Pathology of dentine caries

## *Macroscopic picture (Clinical picture of dentine caries)*

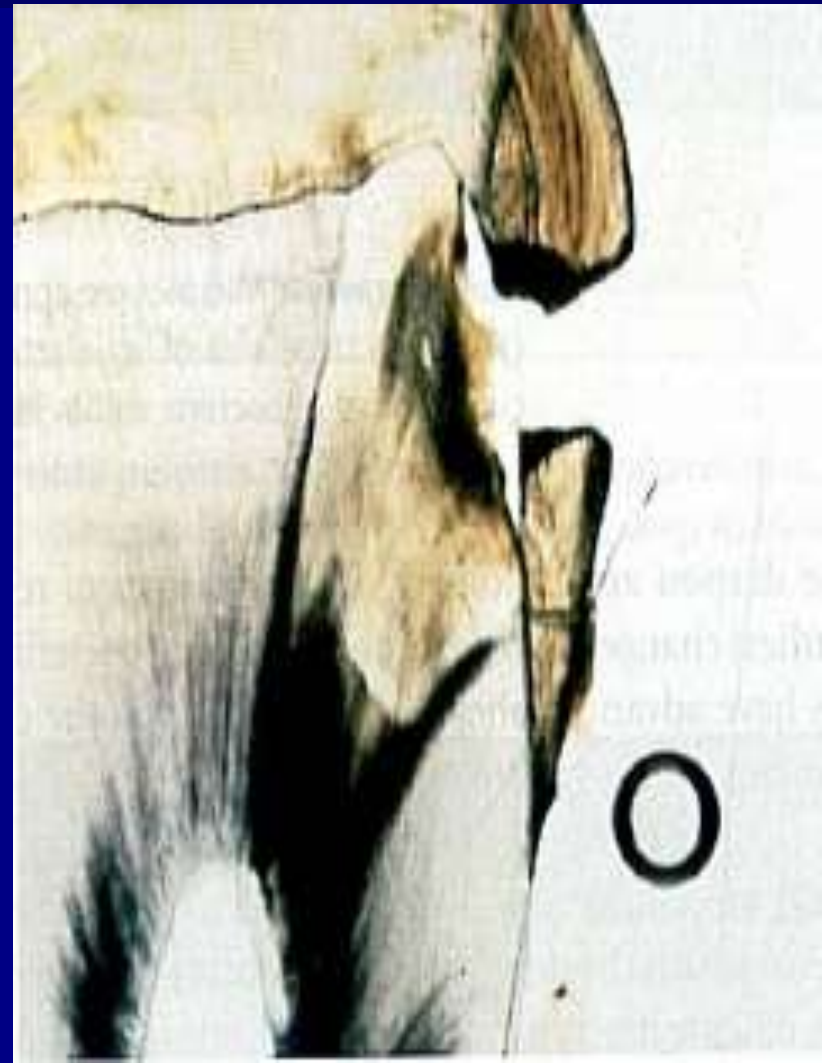
1. The outline of lesion is cone shape with the base toward the ADJ and the apex toward the pulp.

2. Dentine appears to be brownish and is soft to the probe.



**Dentine caries,  
ground section**

# Dentine caries



# ***Microscopic picture***

1. Initial uninfected lesions (prior to cavitations of enamel)
2. Infected lesion
3. Area of destruction



# Initial uninfected lesions (prior to cavitations of enamel)

1. The advancing front of enamel caries has reached the ADJ but the enamel surface is still intact and no cavity has formed.
2. Bacterial products (acids and enzymes) thus reach the ADJ and cause the initial uninfected dentine lesion, which **is characterized by the following zones:**

# Initial uninfected lesions (Dentine zones)

## *1. Zone of reactionary or reparative dentine formation (Zone 1)*

- This zone may be due to its irritation by acid.
- It precedes any dentinal changes.
- The new dentine is formed at the pulpal end of the lesion.
- The dentine may be regular or irregular.

## *2) Zone of sclerotic or translucent dentine formation (Zone 2)*

## *3) Body of the lesion (Zone 3)*

- The body of the lesion is characterized by the presence of dead tracts.

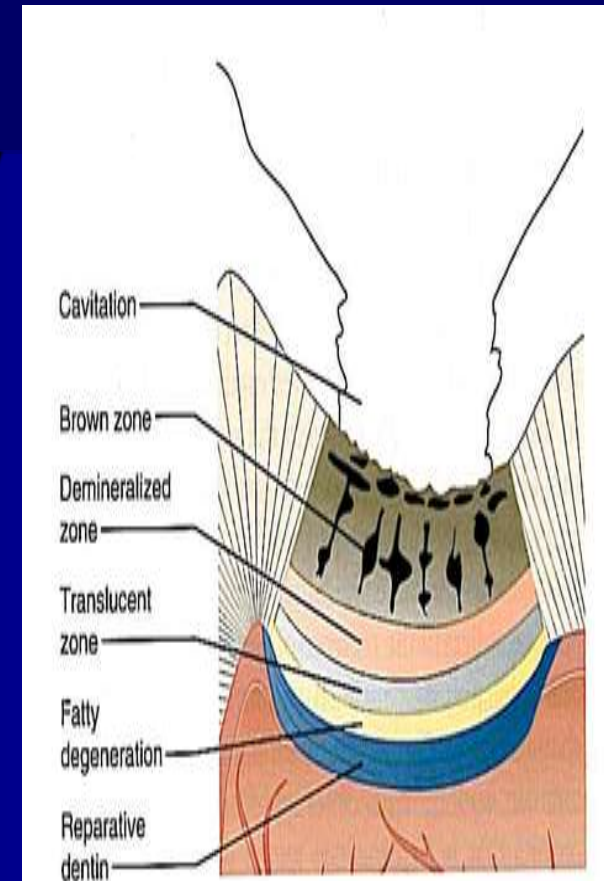


FIGURE 3-9

**Dentin caries.** Microscopic zones of an advanced carious lesion in dentin.

# ***Dead tracts***

are dark due to their high content of organic material when ground sections are examined with transmitted light are empty dentinal tubules containing air and degenerated odontoblastic processes.

- ***The pulpal end of a dead tract*** is occluded by a thin layer of secondary dentine to prevent microorganisms from reaching the pulp.
- ***The superficial part of these dead tracts*** (near the source of the acid) shows decalcification.



## Infected lesion

- This develops after bacteria invasion of enamel and formation of an enamel cavity
- Decalcification involves the wall of the dentinal tubules allows microorganisms to become packed and fill the tubules with pure form of bacteria. E.g. bacilli or cocci. These are called *(Pioneer bacteria)*.

These bacteria penetrate the dentinal tubules before any clinical evidence of caries.

# (Dentine zones)Infected lesion

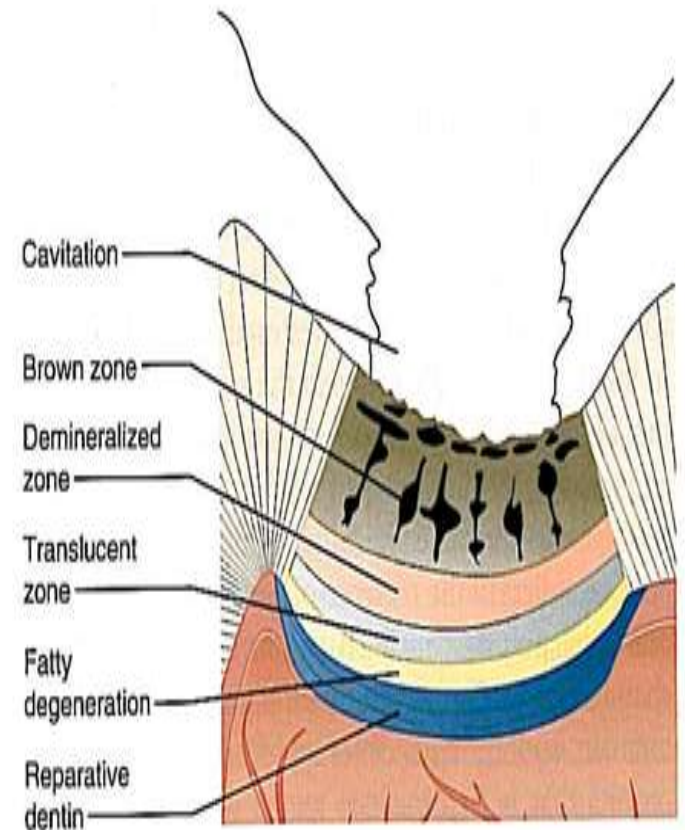
*The lesion consists of 4 zones which are:*

**Zone 1:** Mild pulpal inflammation

**Zone 2:** Reparative or reactionary dentine

**Zone 3:** Sclerotic (Translucent ) dentine

**Zone 4:** Body of the lesion



**FIGURE 3-9**

**Dentin caries.** Microscopic zones of an advanced carious lesion in dentin.

# Body of the lesion (*Zone 4*)

**zone 4 consists of:**

**1. Decalcified uninfected zone**

It is the deepest zone showing decalcification but without the presence of bacteria.

**2. Decalcified infected zone**

*This shows the following zones:*

**1. Pioneer bacteria:** which are usually cocci or bacilli

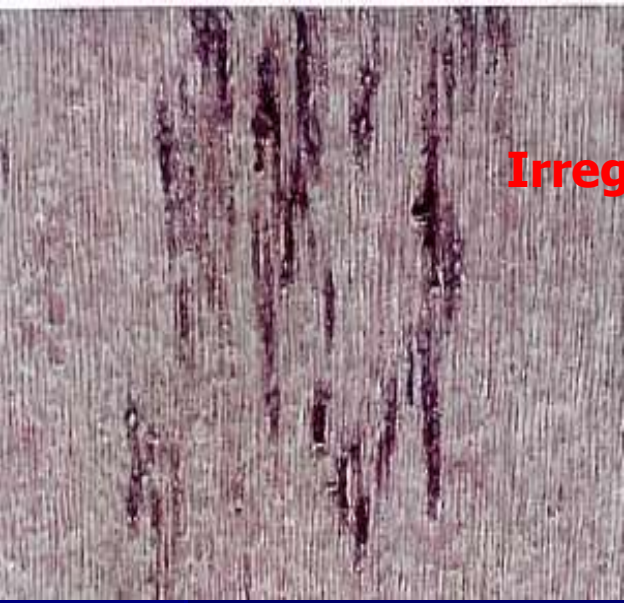
**2. Beading dentinal tubules:** which is lateral distension of the dentinal tubules by the action of the proliferating microorganisms.

**3. Liquefaction foci:** which are formed by focal coalescence and breakdown of dentinal tubules. These are ovoid foci of destruction filled with necrotic debris and parallel to the course of tubules.

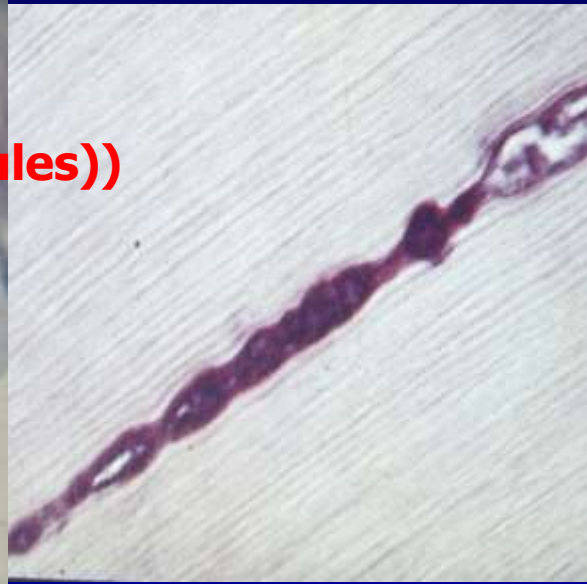
**4. Transverse clefts:** which are formed at right angle of dentinal tubules due to extension of caries along the lateral branches



# Late dentine caries



Irregular beading of dentinal tubules))

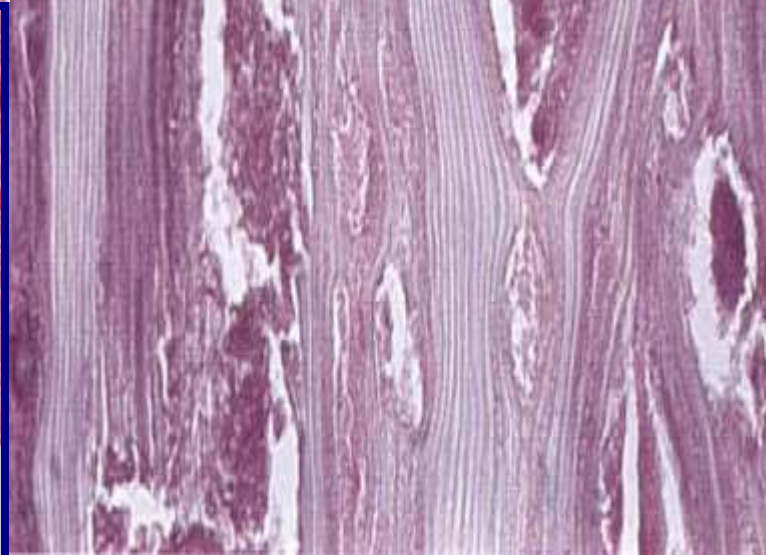
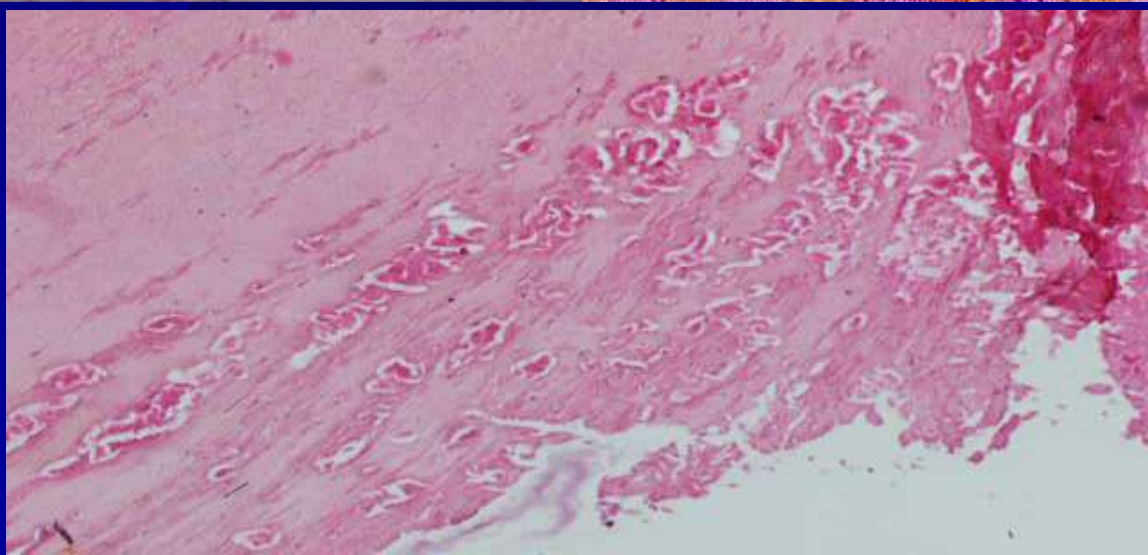
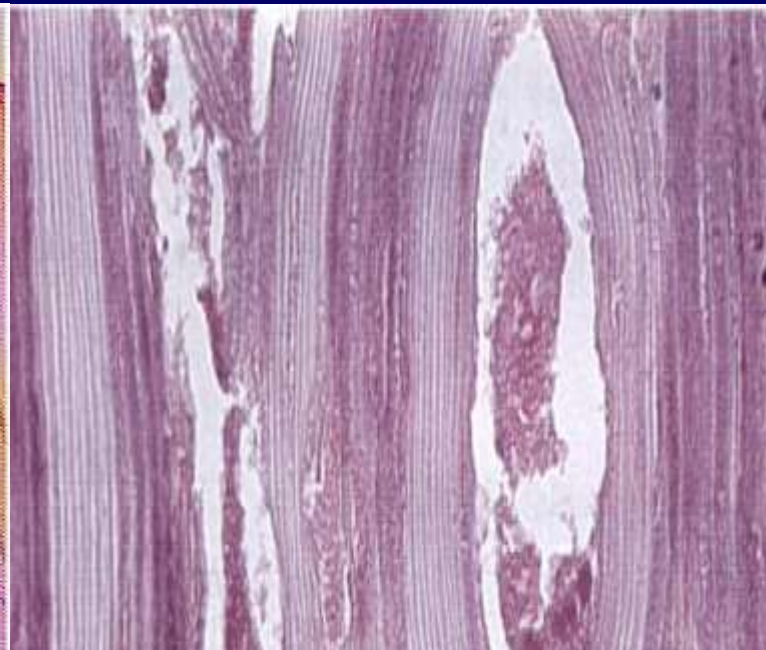
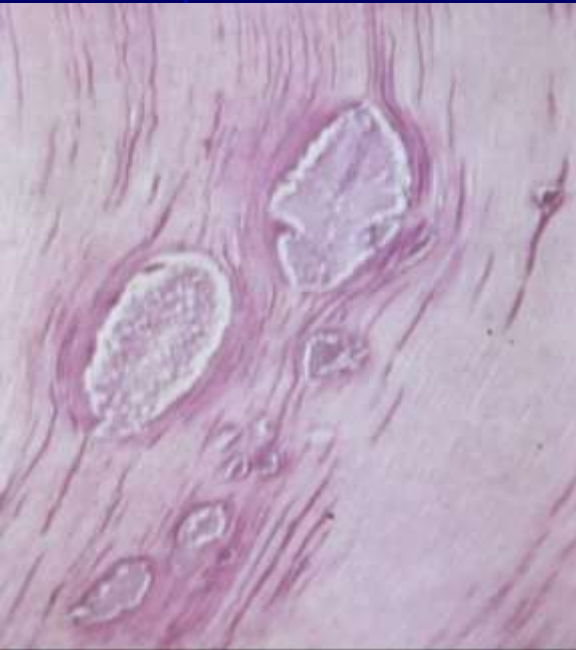


transverse cleft





# Late dentine caries ( liquefaction foci )



### **III. Area of destruction**

It occurs due to destruction of the organic matrix after removal of dentine minerals.



A scenic mountain landscape. In the background, a large, rugged mountain peak is covered in patches of snow and ice, with some rocky outcrops visible. The sky is blue with a few wispy clouds. In the middle ground, there are several tall, thin trees with green foliage. In the foreground, a green field is filled with small yellow wildflowers. A small, dark wooden building with a gabled roof is visible on the right side of the field.

# **Pathology of root caries**

# Pathology of root caries

- It begins with formation of dental plaque having gingival recession in old patients.
- Microorganisms invade cementum along Sharpey's fibers and since  
cementum is formed in concentric layers,  
microorganisms spread laterally  
between layers





**dental caries  
prevention**



# Methods of caries control

## *These methods include:*

1. Substances alter tooth surface, e.g. fluorine. This is done by fluoridation of water supply
2. Substances interfere with carbohydrate degradation, e.g. vitamin K.
3. Substances which interfere with bacterial growth, e.g. ammonium Schlorophyll and penicillin compound.



# Evaluation

الحمد لله

# True or false

(1) Enamel caries occurs in 3 phases (      ) .

(2) In smooth surface enamel caries, the carious lesion is cone-shaped, with the base of the cone at the apex pointing towards ADJ and the apex pointing towards enamel surface (      ) .



# MCQ

( 1) **Increase of caries incidence due to :**

1. No amonia in compositon in saliva.
2. Decrease in amount of saliva only.
3. Thick, mucinous saliva and Low of PH.
4. All of the above

(2) **To reduce caries incidence:**

1. increase Vitamin D and decrease carbohydrate .
2. Decrease Vitamin D, and decrease carbohydrate.
3. Decrease Vitamin D and carbohydrate.
4. increase Vitamin D and carbohydrate.

(1) Dental caries may be due to several contributing factors. These factors are.....,.....,....., .....

(2) Caries may progress faster in hypoplastic teeth and or ..... teeth if compared to normal teeth.

(3) ..... as a type of carbohydrates give more caries as they are easily break down, diffuse rapidly into ..... and they are most frequently used.

(4).....carbohydrates increases caries as they remain attached to the tooth.

(5) ..... is important in the etiology of caries because acid is generated within its substance.

(6) Composition of dental plaque is.....,  
...../...../.....

(7) plaque formation can be prevented by  
...../ ...../ .....

(8) .....is salivary glycoprotein which deposited, and then colonization of bacterial flora takes place.

(9) ..... is sticky, gelatinous substance that enables the microorganisms to adhere to the tooth and form the plaque that initiates caries in smooth surface.



# Thank you

عندي امل في بكرة



